IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TEXAS
TEXARKANA DIVISION

THE STATE OF TEXAS, Plaintiff VS. THE AMERICAN TOBACCO COMPANY; R.J. REYNOLDS TOBACCO COMPANY; BROWN & WILLIAMSON TOBACCO CORPORATION; B.A.T. INDUSTRIES, P.L.C.; PHILIP MORRIS, INC.; LIGGETT GROUP, INC.; LORILLARD TOBACCO COMPANY, INC.; UNITED STATES TOBACCO COMPANY; HILL & KNOWLTON, INC.; THE COUNCIL FOR TOBACCO RESEARCH-USA, INC. (Successor to Tobacco Institute Research Committee); and THE TOBACCO INSTITUTE, INC., Defendants	CIVIL ACTION NO. 5-96CV91 UNITED STATES JUDGE: DAVID FOLSOM UNITED STATES MAGISTRATE: WENDELL C. RADFORD

DEPOSITION OF
MICHAEL SPEER, M.D.

DEPOSITION OF MICHAEL SPEER, M.D., taken on the 3rd day of September, 1997, at Methodist Hospital, 6565 Fannin, Conference Room I, Houston, Harris County, Texas, between the hours of 9:10 a.m. and 5:01 p.m., pursuant to Notice and stipulation of counsel.

I N D E X

E X H I B I T I N D E X

Please note: In day two, Volume II, of Dr. Speer's deposition, it was noted that Exhibit No. 11 could not be found with the other exhibits.

A P P E A R A N C E S

COUNSEL FOR THE PLAINTIFF:

PROVOST UMPHREY LAW FIRM, L.L.P.
2901 Turtle Creek Drive
Suite 250
Port Arthur, Texas 77643-2307
By: Mr. Bryan Blevins

COUNSEL FOR THE DEFENDANTS, LORILLARD TOBACCO COMPANY:

THOMPSON COBURN
One Mercantile Center
St. Louis, Missouri 63101
By: Mr. Michael B. Minton

ALSO PRESENT: Mr. David McCarble

THE VIDEOGRAPHER: It's September the 3rd, 1997. The approximate time is 9:10 a.m. We're on the Record.

MICHAEL SPEER, M.D.,
called as a witness, was duly cautioned and sworn by the Court Reporter to testify the truth and nothing but the truth, and thereupon in answer to questions propounded by counsel, testified as follows:

EXAMINATION

QUESTIONS BY MR. MINTON:

Q. Good morning, Dr. Speer.
A. Good morning.
Q. Let me reintroduce myself. My name is Mike Minton. I represent Lorillard Tobacco Company. And we're here today to take your deposition in a case entitled The State of Texas versus American Tobacco Company, et al. And I'd like to just start by asking you a few preliminary questions.
First of all, let me -- let me just ask that if I ask you a question that -- that you have a problem understanding, will you tell

me and I'll try to rephrase it so that we can be sure that when you have answered a question it's one that you've understood?
A. Certainly.
Q. Thank you. Have you had occasion to have your deposition taken before?
A. Yes.
Q. Can you give us an approximation of how many times?
A. Over the last 16,17 years, probably six, seven, maybe.
Q. Okay. Have those been generally in the context of medical malpractice-type suits?
A. True.
Q. All right. And have you been appearing as an expert witness in those cases?
A. Yes.
Q. All right. And have -- have you appeared more for the plaintiff, more for the defense?
A. So far as depositions it's been almost all for the defense.
Q. All right. How about trial appearances? Have you made any of those?
A. Three.
Q. All right. In -- in the same context,

medical malpractice cases?
A. Yes.
Q. All right. Again for the defense?
A. Yes.
Q. All right. Have there been any lawsuits that you've testified in that have been anything other than malpractice cases?
A. Yes.
Q. What were they?
A. There were two regarding withdrawal with privilege without due process.
Q. All right. Those were cases involving the administration of -- of the hospital?
A. Yes.
Q. All right. And this particular hospital where we are?
A. No.
Q. All right. What -- what hospital was that?
A. I can't remember.
Q. What city were the cases held in?
A. One was, I think, brought in the Bryan/College Station area in Texas. And the other was northeast of Los Angeles in California.
Q. All right. And were -- was your testimony in

the context of defending the hospitals' actions?
A. No.
Q. All right. Was it -- was it -- well, why don't you just tell us rather than me trying to figure it out.
A. Well -- and -- as I said, both instances where -- were where hospitals were proposing to withdrawal or limit medical practice of an individual practitioner without due process.
Q. All right. And in what context did you testify in those cases?
A. For the practitioner.
Q. All right. Saying that the -- that the hospital should not have terminated their privileges under the circumstances?
A. Correct.
Q. All right. Do you recall the names of either of those cases?
A. No.
Q. All right. Do you recall the names of any of the lawyers involved in those cases?
A. No.
Q. What approximate dates are we talking about here? A long time ago?

A. Yes, relatively speaking. I think one was maybe five years ago. And the other one was longer than that.
Q. All right. Was the one five years ago down in College Station?
A. Correct.
Q. All right. If -- if you need or want to take a break at any time today, just say so and we'll go ahead and take a break. Whether you're getting beeped or just --
A. That's fine.
Q. -- want a comfort or convenience break, just say so. This is not a test of endurance.
Let me start out by asking you how you came to be involved in this case.
A. A colleague of mine at the MD Anderson Hospital approached me and asked whether I would be willing to serve as an expert for the State of Texas.
Q. Was that Dr. LeMaistre?
A. No.
Q. Who was it?
A. I knew you were going to ask that. And my memory for names is absolutely atrocious. He's a radiologist over there. It will come

to me probably sometime during the course of the day and I will be happy to tell you when my -- when my synapse is functioning.

Q. All right.

(Two unidentified ladies enter the room)

MR. MINTON: Joe, you could have -- thank you.

UNIDENTIFIED SPEAKER: We just brought it ourselves.

MR. MINTON: All right.

UNIDENTIFIED SPEAKER: Women power, huh?

MR. MINTON: Absolutely. Thank you very much.

UNIDENTIFIED SPEAKER: Okay. You-all have a good afternoon.

(Two unidentified ladies exit the room)

Q. (By Mr. Minton) Approximately what time frame are we talking about?

A. Probably April -- March-April of this year. May, maybe.

Q. All right. And do you remember the substance of the conversation you had with this

colleague?
A. Yeah. I just related what the substance was.
Q. That -- well, I -- if so, I guess I missed it because I gathered that what you told us was that you had been approached by a colleague who was a radiologist, whose name you can't recall, about participating in a lawsuit.
A. Correct.
Q. All right. Was there any --
A. On the -- for the State of Texas.
Q. On behalf of the State?
A. Correct.
Q. All right. Did he describe what the lawsuit was about?
A. It was common knowledge that the State of Texas was bringing suit against the tobacco companies.
Q. All right. And did you have an understanding then about what the nature of the State of Texas' claim is?
A. I'm not too sure I understand your question.
Q. Do you -- do you understand the reasons why the state is -- is bringing the action and what it is they're seeking to recover in the case?

A. My understanding is that they're seeking to recover monies expended on the treatment of patients that are covered under federally mandated programs such as Medicare and Medicaid.
Q. All right. And do you have an understanding of the legal basis upon which the state is pursuing this claim?
A. Not being a lawyer I have no understanding of the legal issues in that regard.
Q. Okay. Do you have any sort of layman's understanding about what it is that the state thinks entitles it to recover from the tobacco companies?
A. That's between you and the state lawyers. I'm not here to have an opinion on that.
Q. Okay. About how long did this conversation last?
A. A minute.
Q. Okay. Was it a telephone call or just...
A. No, just stopped me in the street.
Q. All right. Did -- did this colleague mention the name of any lawyers who were participating in the case?
A. No.

Q. All right. What -- did you take any action on the basis of that conversation?
A. No.
Q. Were you then contacted by somebody?
A. Correct.
Q. And who contacted you?
A. A Ms. Suzanne Klok, I believe.
Q. Okay. Could you spell that last name for us?
A. K-l-o-k.
Q. Okay. And who is Suzanne Klok?
A. I don't know. Her office was at that time -- at least in May 16th of this year -- at 2901 Turtle Creek Drive, Suite 201, Community Bank Building, Port Arthur, Texas.
Q. All right. You're reading from a document. May I have a look at what you're reading from?
A. Certainly.
Q. Thank you. All right. This refers to a -- this letter which we'll go ahead and mark as Exhibit 1 to your deposition.
THE COURT REPORTER: Can you excuse me just for one moment?
MR. MINTON: You bet. Let's go off the Record.

THE VIDEOGRAPHER: The time is 9:18. We're going off the Record.
(Discussion off the Record)
(Speer Exhibit No. 1 marked for identification)
THE VIDEOGRAPHER: The time is 9:19. We're on the Record.

Q. (By Mr. Minton) Dr. Speer, we've marked the letter that -- that you were referring to just a moment ago as Exhibit 1. And in it there is a reference you make as the author of the letter to some previously discussed issues.
A. Right. She called me and asked if I was willing to submit a letter outlining my opinion regarding the effect of tobacco in babies. And I said I would, and I did.
Q. All right. Is that a summary of a longer conversation or is that --
A. That is essentially it.
Q. Okay. And did she indicate to you in what context that report was going to be used?
A. I presume within the context of my serving as an expert for the State of Texas in this particular lawsuit.

Q. Did she ask you to do that?
A. Yes.
Q. All right. Did you agree to do that?
A. Yes.
Q. All right. All right. Have you been retained in some way?
A. I'm not too sure I understand.
Q. Is there any kind of agreement or understanding that you have with the State of Texas regarding your appearance as an expert witness in this case?
A. You're going to have to be clearer.
Q. Well, have -- in the -- in the cases in which you've been retained as an expert for the defendant, have you been retained in those cases?
A. What do you mean by retained?
Q. Do you have some sort of understanding regarding the terms of your engagement as an expert witness?
A. I'm still not quite sure what you're asking. I charge -- I charge for what I do. I submit bills for what I do. I have not received any nor do I ever receive money up front as a retainer.

Q. Okay.
A. Which is where I think you're going.
Q. I asked an unclear question, and you did exactly the right thing in pointing it out to me that it was unclear. Let me -- let me try to break it down and make it clear.
Do you have any sort of agreement with the State of Texas regarding any compensation in this case?
A. You mean, for example, a signed contract?
Q. No. Any -- any kind of understanding or agreement which would include, the way I'm phrasing the question, talking to somebody about it over the telephone.
A. The only "agreement" that I have is with this gentleman's law firm that I will bill his firm for what I do.
Q. Okay. That's Mr. Blevins that you're talking about and Provost Umphrey is the firm?
A. Correct.
Q. Okay. And what is the agreement with respect to what you're going to bill the state for -- for your work in this -- in this case? Is there an hourly rate for that?
A. Yes. It's an hourly rate.

Q. And how much is that?
A. For what?
Q. For your work per hour.
A. Depends on what I'm doing.
Q. Okay. Why don't you tell us what the different rates are depending on what you're doing.
A. I'd be happy to. $300 an hour for review of documents and literature including the generation of a report; $500 an hour for deposition and trial. If I have to go somewhere for the day, we'll work that out.
Q. Is that the same rate that you charge for working malpractice cases?
A. Exactly the same.
Q. Okay. What is the reason why there is a difference between the hourly rate for reviewing?
A. Because in depositions and trial I get to meet with people like yourselves.
Q. What does that mean?
A. That means that on face-to-face it takes more of my energy and thinking when I'm being cross-examined, either in a deposition or a trial environment.

Q. The -- the other issues, if any, that were discussed with -- with Ms. Klok were what?
A. I think I've stated what I discussed. She asked me to write a report, a brief report, regarding my opinions regarding tobacco effect on babies.
Q. All right. Did she -- did she retain you in that conversation or did she say, "We want you to testify on behalf of the State of Texas"?
A. I'm not too sure if she did or not.
Q. Okay. When was the first announcement that you had that -- that the State of Texas was going to ask you to testify in a deposition?
A. The State of Texas has never asked me to testify.
Q. Someone representing the state.
A. Someone from his firm asked me to -- reconfirmed whether I would be an expert witness and testify in this lawsuit.
Q. All right.
A. I said yes.
Q. And -- and about when was that?
A. In and around the time I generated the report.

Q. Okay. Well, before or after?
A. I don't know.
Q. Okay. Have you made any notes in connection with your work on this case?
A. The report.
Q. All right. Is the report the only time that -- that there have been written words generated by you in connection with your work on this case?
A. I think so.
Q. In other words, there are no notes, for instance, of any literature that you've reviewed for this case?
A. No.
Q. All right. There are no notes of any documents other than medical literature that you've reviewed for this case?
A. There are no notes.
Q. Okay. About how much time have you spent overall working on this case so far?
A. We haven't calculated it up. I haven't even submitted a statement as of yet. Probably around 15 hours; 13 to 15, in that ballpark.
Q. All right. And what have those hours been spent doing?

A. Mostly reviewing transcripts and a couple -- about two and a half hours in toto meeting with this gentleman and his colleague.
Q. All right. Do you know who Mr. Blevins' colleague was that was at the -- at the --
A. He was a tallish, thinner gentleman and with darker complexion than Mr. Blevins. As you can gather, I'm terrible on names.
Q. Okay.
A. I'll tell you a little story. I was dating my wife some 20 plus years ago. I wasn't dating anyone else, and I had to have her name written by the telephone so that I would say her name correctly. And it's a very difficult name. It's Mary.
Q. Okay. The transcripts that you've reviewed, can you tell us who were the witnesses that were being deposed?
A. Some were reviewed in more depth than others. Let's see. There was a Dr. Carpenter who I reviewed in the most depth. There was a Dr. Moody, I believe, that I briefly reviewed. There was a transcript, I think, from another action in another state, I think a Dr. Sachs, that I briefly reviewed. And

there was a -- one from a -- I think a neonatologist in the state of Arkansas that I briefly reviewed. And there are undoubtedly others.

Q. All right.

A. But those are the names that come immediately to mind.

Q. Are -- are those depositions in your possession?

A. They are over on the table.

Q. Okay. The -- did you make any notes?

A. No.

Q. On those?

A. No.

Q. What was the purpose of the review of -- of the depositions?

A. Get a sense of what questions were being asked the individual witnesses.

Q. All right. And who provided you with the depositions?

A. Mr. Blevins' firm.

Q. All right. His idea or yours?

A. His.

Q. Okay. And when -- when did you review those?

A. Bits and pieces from the first time they came

until I -- actually, the night before last.
Q. And who was it that you reviewed the night before last?
A. Dr. Carpenter's.
Q. Okay. What -- what are your opinions or feelings with respect to your review of the Carpenter deposition?
A. It's a deposition.
Q. All right. Well, are there -- are there things that stand out in terms of the work that you've done in the case, things that he said with which you agree or disagree?
A. No.
Q. Do you find yourself in substantial agreement with what Dr. Carpenter has said?
A. Many of the things that he pointed out, I think he pointed out correctly.
Q. All right. Do you find yourself in significant disagreement with Dr. Carpenter with respect to -- to any of his testimony?
A. Well, I'd probably have to -- we'd probably have to go page-by-page in order to really come down and see if there is anything I have signficant disagreement with.
Q. Okay. But as you sit here today, I mean,

there's nothing, having reviewed it night before last, that you -- you say, "No. I -- that's just glaringly wrong"?

A. Not that I remember.

Q. Okay. How about the other -- the other doctors whose depositions you reviewed?

A. I don't even really remember the exact content of those depositions.

Q. Okay. And --

A. I'd be happy to review any portion thereof, if you'd like.

Q. The -- of the 13 to 15 hours total that you've spent, how much would you say has been involved in the review of the depositions?

A. Remember we're talking since May.

Q. Right.

A. Okay. And it's been bits and pieces of time over that period of time to today. Probably we've taken two and a half hours for Mr. Blevins and colleague. Probably an hour, hour and a half to maybe even two hours to, you know, do a quick review of what I reviewed in order to come up with -- in the composition of the report. So at the most, I'd say we're talking four and a half. So

13, roughly, minus four and a half is --
Q. Eight and a half?
A. Eight and a half over a period of May, June July, August. Two hours a month, roughly.
Q. Did -- did anyone ask you to look at particular issues? Say that the -- that they anticipated that -- that these issues would be highlighted or emphasized?
A. No.
Q. Okay. Have you been given any company documents? And by that, I mean any -- any internal tobacco company documents.
A. No.
Q. Okay. Have you been provided with any medical journal articles?
A. Yes. I was provided by -- with one that I already had.
Q. The Drewes article?
A. Right. I had already read that. My copy was clearer than the copy provided, so I kept mine.
Q. Okay. Do you know Caroline Drewes?
A. No.
Q. All right. Do you review for that journal?
A. Pediatrics?

Q. Yes.
A. Uh-huh.
Q. Did you review that article?
A. No.
Q. Do you know who did?
A. No.
Q. What article -- or excuse me -- what journals do you -- do you currently review for?
A. The New England Journal, Pediatrics, Journal of Pediatrics, Journal of Pharmacy and Therapeutics. I have reviewed for ACTA Scandinavia Pediatric. And I think that's about it.
Q. Okay.
A. It's in my CV.
Q. How about --
A. You do have a copy of my CV.
Q. I do. Okay.
A. They're listed.
Q. Yeah. It's just that as I gather sometimes those change from time to time. And I wanted to get an idea of -- of currently which ones you're reviewing for. How does that work? How does one become a reviewer for a --
A. Good question.

Q. -- publication?
A. Good question.
Q. All right. Do up know people on the editorial board, for instance, of The New England Journal of Medicine?
A. No.
Q. You know Marsha Angel, for instance?
A. I know of her. I don't know her.
Q. Okay. Have you read any of her works?
A. Mostly editorial comments.
Q. All right. Do you have any idea how you became a reviewer for The New England Journal of Medicine?
A. No. Presumably, somebody recommended that I knew something about an article that they wanted to send to somebody and they sent it.
Q. Okay.
A. And asked me to review, and I said, "Okay. I will review." And I sent the review back.
Q. Is it your --
A. And they said thank you.
Q. Is it your understanding that reviews -- and I'm probably going to use a poor word here, but it's just my limited vocabulary -- are episodic or are they -- do they occur as a

result of some methodological process of the -- of the publication? And by that, I mean are there -- is it your understanding that -- that publications have a stable of reviewers or a -- or a -- a group of people who are their reviewers for particular areas and they'll -- they'll give them assignments on some kind of planned basis or -- or does the reviewing assignment come in some other way?

A. Not being a member of the editorial board of any of those publications, I can't tell you how they select any given reviewer. From the receiving side of papers, my presumption is that the papers that I've reviewed usually have something to do with an area of research that I've published in.

Q. All right. And -- and what area is that?

A. It's fairly eclectic.

Q. What areas?

A. Vitamin E in interventricular hemorrhages; CPK, creatine phosphokinase also within the area of interventricular hemorrhages; infections in the newborn, both bacterial and some viral. Those are the major areas.

Q. And -- and not -- if this isn't an un -- if this is -- if this is not an unfair synopsis, say so. But they deal basically with interventricular hemorrhage and bacterial and viral infections of the newborn?
A. At the moment, yes.
Q. Okay. Have there been other areas in the past that have been areas in which you've found yourself reviewing articles?
A. Those are the main.
Q. Okay. How many articles -- say over the last five years, how many articles have you been asked to review?
A. I can't really say. I'm -- I'd be guessing. Six or seven, probably. It's not a huge volume.
Q. Six. So one to two a year is a pretty good --
A. Yeah.
Q. Okay. Do you remember, for instance, what the last article was you reviewed for The New England Journal?
A. No.
Q. Any idea how long ago it's been?
A. Six months.

Q. But as you sit here, you don't have any recollection of what the article was? Has it appeared in print? Do you know?
A. I recommended rejection.
Q. Okay. Why was that?
A. Because it was an inappropriate article for The New England Journal.
Q. In what way?
A. It was a case report, basically. Had no control trial. It wasn't randomized. It was a case report.
Q. Didn't stand up to -- to epidemiologic scrutiny in terms of -- of presenting some sort of --
A. Well, it's not epidemiologic scrutiny. It just wasn't an appropriate article for The New England Journal of Medicine. It's a case report. And The New England Journal rarely publishes case reports.
Q. All right. What -- in -- in your view, what is the value of a case report?
A. A case report is an instance that may or may not be unique. It may or may not have relevance to a larger issue. Most case reports are relatively unique and serve to

inform the reader that in this particular type -- in this particular patient this occurred, and the potential of that phenomenon occurring in other patients is probably real and that you need to be aware of that.

Q. All right. Are there particular types of publications where -- where a case report is more appropriate?

A. Yeah. The Journal of Perinatology would probably be more appropriate with case reports. Usually not single case reports but a several, say two or three or four or five cases, will appear in the Experience and Reason section of Pediatrics.

Occasionally a short article that's close to a case report may occur -- or may appear in the Journal of Pediatrics. Southern Medical Journal will have some case reports. Rarely will JAMA have case reports. It depends on the -- on the journal.

The authors of medical articles usually pick the journal that they think will accept their work. Sometimes they pick a journal on the topside that they don't think they can

get in; but if they can, it will be great. But then they take the article and move it to a journal that will be more likely to publish it.

Q. All right. Do you subscribe to the -- to JAMA?

A. Yes.

Q. All right. Did -- did you happen to see Stan Glantz' article in JAMA that dealt with tobacco industry documents?

A. Possibly.

Q. All right. But as you sit here, you have no recollection of it?

A. Not specifically, no.

Q. All right. Do you -- can you see how an article dealing with Dr. Glantz' interpretation of tobacco industry documents would -- would comport with the -- the editorial policies of JAMA insofar as the types of reports that JAMA publishes?

A. Not -- not remembering exactly what was in the article, I fear I can't answer your question.

Q. All right. Do you know who Dr. Glantz is?

A. No.

Q. Have you been provided any documents that have been -- that you've given back; in other words, that you haven't kept in connection with your work on this case?
A. No, I don't think so.
Q. So everything that you've been provided is -- is sitting over there with the box?
A. It's possible there's something on top of my desk. And if you saw the top of my desk, you'd understand what I'm saying.
Q. Okay.
A. But not consciously withheld.
Q. Top of the desk I consider to be sort of like home free when you're playing games as a kid. It just sort of doesn't count given the circumstances.
Is this the -- is Exhibit 1 the only letter that's been exchanged to or from counsel in this case in connection with the --
A. Oh, they've sent little cover letters with the documents.
Q. Just transmittal memos of what's inside?
A. Correct. Here. Here's another hundred pages or thousand pages for you to look at in your

spare time.
Q. Okay. You mentioned some depositions. Have there been any other reports, witness reports or witness statements, or anything like that that you've reviewed?
A. Can you stop for just a moment and let me --
Q. You bet.
THE VIDEOGRAPHER: The time is -- the time is 9:39 a.m. We're going off the Record.
(Discussion off the Record)
THE VIDEOGRAPHER: The time is 9:40 a.m. We're on the Record.
Q. (By Mr. Minton) Dr. Speer, you were kind enough to retrieve for us some bound volumes. One entitled Robert J. Carpenter, M.D., defendant expert witness; Robert W. Arrington, M.D., defendant expert witness; Percy Luecke, M.D., defendant expert witness; and Robert Woody, M.D., defendant expert witness. And we'll mark those as Exhibits 2, 3, 4 and 5 respectively.
Are these reports of witnesses that you've reviewed as well?
A. I've glanced at those, the contents of those.

Much of them I did not review in depth.
MR. MINTON: All right. Would you gone ahead and mark those, please?
(Speer Exhibit Nos. 2 through 5 marked for identification)
MR. BLEVINS: Just for clarification, those are the disclosures that were previously made. They were just bound, but those are the same.
MR. MINTON: You can go ahead and put that on the Record.
THE COURT REPORTER: Okay.
MR. MINTON: We'll wait until you're done marking. You can't do two things at once.
THE COURT REPORTER: I'm sorry.
THE WITNESS: You can't? I thought court stenographers could do multiple things at once.
MR. MINTON: Well, that's true. They have to take two people down at once. I know that. It causes them some degree of frustration.
THE WITNESS: Only if we talk at the same time.

THE COURT REPORTER: Do you need these or --

MR. MINTON: I'm just going to ask him a couple more questions about those.

THE COURT REPORTER: Thank you.

MR. MINTON: We're still off -- we never went off.

Q. (By Mr. Minton) Okay. Dr. Speer, I'm going to ask you essentially the same question I asked you about the depositions. Having reviewed these, with your qualification that it was a brief review, was there anything that stood out in terms of sensing disagreement with what these witnesses said in their reports?

A. Given that my review was a number of months ago and relatively brief at the time, I would have to ask you to point out certain areas within those that you want me to comment on. I really can't comment on all four of the documents in a global sense.

Q. Okay. But there -- for instance, there wouldn't be any markings that you've made or note --

A. No. I don't make markings, and I did not

take notes. But if you have a certain area in those -- what are they called?

MR. BLEVINS: Disclosures.

A. -- disclosures that you wish me to address, I'll be more than happy to do so.

Q. Thank you.

MR. MINTON: Would you mark these 6, 7, and 8, please?

THE COURT REPORTER: Sure.

MR. MINTON: Thank you.

(Speer Exhibit Nos. 6 through 8 marked for identification)

MR. MINTON: Thank you. And, actually, before we start going through those, the two articles that were attached to the disclosure statement, the Drewes article and then the American Academy of Pediatrics, do we have relatively clean copies of those available? Do you know? Because the -- the ones that came through on the fax are -- are pretty difficult to read and I'd like to mark them, but I'd like them to be readable if we do that.

MR. BLEVINS: Yeah. That will -- I mean, if you'd like to break -- I mean, do

you want to mark these now and just take them out of my --

MR. MINTON: If you wouldn't mind.

MR. BLEVINS: Paralegal's going to want to know what the heck I did with them.

(Speer Exhibit Nos. 9 and 10 marked for identification)

Q. (By Mr. Minton) Just to sort of continue with the housekeeping here, Dr. Speer, what I'd like to show you are Exhibits 6 through 10, which I'll represent to you are the disclosure materials that we received from the counsel for the State of Texas with regard to your appearance as an expert witness in this case. And what I'd like to do is look at each one sort of in turn and let's describe what is in there. You have Exhibit 6.

Okay. All right. Is Exhibit 6 your -- a transmittal letter containing your expert report and your CV?

A. Correct.

Q. All right. And the expert report, is it the same as the -- as the report attached to the May 16th, 1997, document?

A. No.
Q. Are there changes that have been made there?
A. The lawyers apparently took the document that I provided them and added some phraseology and then submitted it back to me for approval. I reviewed it. Said, "Fine. If you want to say it this way, that's okay with me."
Q. Okay.
A. And they put it into the disclosure document.
Q. Were the types of changes grammar and sentence structure, that sort of thing, or were there substantive areas that were gone into?
A. I'm not -- I don't think there is any really substantive areas, mostly grammar. They like to divide things up into paragraphs.
Q. Okay.
A. I'd hate to see their desk.
Q. All right. Exhibit 7 appears to be a list of your publications. If you could just confirm that for us.
A. Publications, abstracts, papers presented at meetings, and invited -- a list of invited participant at research seminars or meetings,

yes.
Q. 8 gives us some information on books, articles and papers authored by the -- by you which has a reference "see CV." It has a listing of your prior testimony. And I think there is a cutoff date. It's 1994 and forward that applies to this.
A. Mr. Blevins is compulsive. And he wished to have something in the document that reflected the fact that I had given testimony before. And given the fact that my memory is atrocious, as already exhibited, so far as names and dates, because I don't keep a rogues' gallery of that sort of thing, he did some exploring and added a couple of cases that indeed I have -- now, having looked at names, I indeed did testify in those cases.
Q. All right.
A. There are others, but I can't tell you what they are.
Q. Okay. And the basis documents, there are two. And then we've separately marked those. Mr. Blevins was kind enough to give me --
A. Yes. He wanted to have some documents in this area, although I told him that really

there are no single documents or groups of documents that form the basis of my opinion. But he insisted. So I said fine.

Q. What is -- what is the reason for -- is there any reason, other than his insistence, on including these two documents that -- well, I -- maybe I phrased that unfairly. Was it his insistence that these two go in or was it your idea that these --

A. He wanted them in there. I said fine. Didn't make that much difference to me.

Q. All right. Are these -- are these documents of any particular significance to you, Exhibits 9 and 10?

A. No.

Q. Have you -- have you reviewed the -- the Drewes article?

A. Yes.

Q. All right.

A. That formed part of my original report. And as I mentioned earlier, I had actually reviewed that article prior to him speaking with me the first time and presenting the article for my perusal.

Q. All right. How about Exhibit 10? Had you

ever seen that before?
A. That's a relatively old document dating from 1994. And certainly I'm familiar with the academy's stance on tobacco.
Q. All right. But had you seen that particular document before?
A. In all likelihood, yes, because I take Pediatrics and I read it.
Q. Okay. Did you suggest the inclusion of any documents in that list to Mr. Blevins?
A. No.
Q. Okay. As you sit here today, are there any particular articles that -- that you think are noteworthy that you are, in fact, relying on in connection with your opinions?
A. No. It's really the accumulated experience and reading that I've had since approximately 1964 when I started medical school.
Q. And -- and so, as you sit here -- well, no sense in being repetitive.
Had you met or do you know any of the witnesses whose reports, depositions or testimony that you've reviewed?
A. I know Dr. Carpenter.
Q. All right. And how do you know

Dr. Carpenter?
A. Let's see. I'm not too sure whether I knew him in medical school or shortly after he completed his M.D. degree. But I've known Robert at least for 15 or 20 years. He's an obstetrician. He's a high-risk perinatologist and refers patients to our group on a somewhat regular basis because we -- he happens to deliver hish-risk babies at St. Luke's. And we take care of high-risk babies at Texas Children's.
Q. What is your impression of Dr. Carpenter's competence in the field?
A. He's an excellent perinatologist.
Q. All right. Have you written any articles in the area of maternal smoking and health?
A. No.
Q. All right. Have you done any primary research in that area?
A. No.
Q. Have you worked with physicians in some close capacity who were doing primary research in that area?
A. Given the number of physicians that I've worked with over the last 20 plus years and

not knowing everything that those individual physicians have been involved in in terms of research, I probably have worked with a physician that has done some research in the area of tobacco. But I do not know what research that might be.

Q. All right. Have you, in connection with -- let me make my question a little narrower.

In connection with the physicians you're -- you have worked with, you're not aware of any of them, while you were working with them, working on a project related to maternal smoking and health?

A. Correct.

Q. Okay. Do you know Ben Sachs?

A. No.

Q. Do you know what -- in what capacity he testified in the -- in the deposition that you read?

A. I think his capacity was of an epidemiologist statistician. And a lot of the discussion appeared to center on subtle nuances of various population groups. And there appeared to be a lively repartee, if you will, between opinions of whether or not

various and sorted research was done correctly or incorrectly based on the sides that the questions were coming from.

Q. And -- and do you -- having reviewed Dr. Sach's deposition, do you have some overall opinion about the substance of his testimony?

A. If I remember correctly, the substance of his testimony was that many studies have showed that smoking and ill health are related.

Q. And in the spectrum of relationships that can span statistical associations to causal relationships, do you have recollection and/or agreement or disagreement within that spectrum of issues that --

A. Not having been present at the deposition and unable really to ascertain in reading the definition -- the definitions of causes, associations and risks, I really can't say.

Q. What is your own training in epidemiology?

A. As with most physicians who work in an academic environment who do clinical research, I have a working knowledge of epidemiology.

Q. How was it acquired?

A. Experience and reason.
Q. Are you a member of any societies or groups devoted to that discipline?
A. No. Although sections of various societies probably are knowing the breadth of committee structures in various societies.
Q. You're not a member of any subsections of any societies that deal with epidemiologic issues?
A. Not as an announced goal. However, I am a member of the section of peri -- neonatal/perinatal medicine of the academy. They have done recently a survey of its members. That sort of fits into the broad rubric of epidemiology.

Therefore, I am a member of the section that has done some work in that area, but it is not the espoused program of the section, neonatal/perinatal medicine, to study epidemiologic issues, if that answers your question.
Q. What was the member survey that you participated in?
A. It was: Who are you? Where do you practice? How many people do you have in your practice?

How many babies do you take care of in a given year? That sort of thing.

Q. Have you -- have you been asked to review any papers which deal with enunciating correct versus incorrect epidemiologic methods?

A. Are you talking about papers that deal with methodological issues in epidemiology or are you just -- are you asking whether I've reviewed papers in which epidemiology-type of processes were used?

Q. The former. And I'll try and rephrase my question to make it clearer. Have you served as a reviewer of any paper, the focus of which was a proper epidemiologic method?

A. No.

Q. In terms of your experience with epidemiologic methods as a clinician -- and is that -- would that be a fair statement of how that familiarity with the field has -- has developed in your case?

A. Keep -- keep going.

Q. Okay. In terms of your clinical experience with -- with epidemiologic methods, have you come across textbooks that you believe are sound in terms of their enunciation of

epidemiologic principles?
A. Let me rephrase your question and see if I've got it correct. Have I used epidemiologic textbooks to formulate my research?
Q. That would be a portion of what I'm asking. And let's start with that.
A. No.
Q. Have you used epidemiologic textbooks to review the methods that you have designed for your research?
A. Are you including within the definition of epidemiology textbooks, statistical textbooks?
Q. Yes.
A. I have reviewed statistical textbooks and used those.
Q. All right. Which statistical textbooks do you use?
A. I don't remember.
Q. Okay.
A. I can get them for you if you wish.
Q. The -- could you give us the names of -- of epidemiologic texts that you believe are recognized by people who work in the field as -- as being strong, sound and

authoritative texts?
A. I don't think there are any texts of any nature that are authoritative. And I do not know of any particular epidemiological textbooks that are held to be a standard of excellence.
Q. All right. Do you know the names of any?
A. No.
Q. What is your understanding of the term "confounder" as it's used in the epidemiologic vernacular?
A. Not being an epidemiologist, why don't you give me an example.
Q. Well, before I do that, I'd rather have your definition or your understanding of what that term means in the epidemiologic realm.
A. Okay. Are you speaking of confounding variables?
Q. Yes.
A. Okay. A confounding variable -- my understanding of a confounding variable is that it may act with or separate from whatever you're trying to examine as a primary cause or relationship.
Q. All right. Is -- would it be correct to say

that a confounding variable is recognized to be a -- a risk factor for a disease?
A. It may be.
Q. All right. Are there situations in which a -- in something which is recognized to be a confounding variable, with respect to a particular disease endpoint, would not also be a risk factor for that disease endpoint?
A. Could you clarify your question?
Q. If you can tell me what was unclear about it, I'll struggle to do so.
A. The whole sentence.
Q. All right. Can -- are there situations that you are aware of where something that has been recognized to be a confounding variable for the production of a particular disease would not also be a risk factor for the production of that disease?
A. Confounding variables may be -- may be additive or be subtractive regarding the risk of a disease. It can be either one.
Q. They can be -- they can be positively associated or negatively associated?
A. Correct.
Q. All right. And how is -- in terms of

epidemiologic inquiry, how is the effect of confounding either controlled for or reduced in an epidemiologic study?

A. You lost me.

Q. All right. Is -- is confounding, if present, a methodologic flaw in an epidemiologic study?

A. It may or may not be.

Q. All right. Under what circumstances might it be?

A. Well, if you were studying lung cancer and you only took children under the age of 10, you wouldn't have very many young -- you wouldn't have many patients with lung cancer because lung cancer rarely occurs under the age of 10. So, obviously, that's a methodologic flaw because age is a confounding variable when you're talking about lung cancer.

Q. All right. And how might confounding not be a methodologic flaw in an epidemiologic study?

A. If you took patients who are 65 years and older and looked at the incidents of lung cancer, that's an appropriate population.

Q. And by patient selection, then have you controlled for the presence of the confounding variable?
A. You can; sometimes you can't.
Q. Was that the import of the example that you just gave us?
A. You asked for the example of negative and positive confounding variables. And I attempted to do that.
Q. Actually, what I was asking was for a situation in which a confounder could be present and yet not constitute a methodologic flaw in terms of --
A. Then it's not a -- then it's not a -- it's not then a confounder. If it's not -- doesn't have any bearing on what you're looking at.
Q. As a general matter, then is it correct to say that -- that if confounding is present it must either be -- well, it must be controlled for in order to prevent the occurence of a methodologic flaw?
A. No. That's not what I said.
Q. All right. And why is that not true as a general statement?

A. Because you have statistical methodology that can obviate the confounder.
Q. By controlling for it?
A. No. By -- well, yes. In certain statistical terms I suppose controlling in that use of controlling is correct.
Q. All right. And what are the -- the principal means in an epidemiologic study by which confounding is controlled?
A. As I've already told you, I'm not an epidemiologist. I am not a statistician. And I would refer you to those individuals to give you that answer.
Q. All right. Are you familiar with bias in terms of its impact on epidemiologic studies?
A. I'm familiar with bias in terms of it being a variable that needs to be examined very carefully in any study.
Q. Would it be correct to say that it, too, is a methodologic flaw that systematically will cause results that depart from the truth?
A. Only if you know that you have systematic bias present. If you do not -- are not aware of any bias, then not necessarily.
Q. But bias equals a systematic flaw, does it

not?
A. I think I've already answered that.
MR. MINTON: Could you go back? He probably did and I just didn't hear it right. Will you read back the --
THE COURT REPORTER: Is it the very last answer?
MR. MINTON: Two -- two ago, I think.
THE COURT REPORTER: Two ago?
MR. MINTON: Yeah.
(Requested portion of testimony was read back)
THE COURT REPORTER: Keep going?
MR. MINTON: No. I think he answered the question. Actually, he was right.
Q. (By Mr. Minton) How does one control for bias in the context of an epidemiologic study?
A. Again, not being an epidemiologist nor a statistician, I'm not qualified really to tell you how to set up an -- a purely epidemiologic study to eliminate all bias, because what you're doing in epidemiologic

studies is observing natural phenomena. You can control for variables to attempt to limit bias and/or you can state in the design of the study or in the results of the study that a bias is there. But there -- as I already said, there may be an unexpected bias that you're unaware of.

Q. Is there any technique that you know of that can control for bias discovered after the fact? And what I'm referring to is the type of bias that you just referred to.

A. Multiple regression analysis.

Q. All right. And do you know how multiple regression analysis seeks to determine the effect of bias in an epidemiologic study?

A. That's why I hire statisticians.

Q. Is -- is a fair answer to that question no?

A. I don't do that type of analysis.

Q. And would it be fair to say that since you don't do that type of analysis you don't know how it works?

A. No. I know in concept that it eliminates -- it examines various variables that may have or may not have an impact upon the null hypothesis. And it either does or does not

eliminate those variables.
Q. All right. The null hypothesis is the -- is the hypothesis that there is no relationship between the dependent and the independent variable?
A. There is no relationship in the issue that you're studying between -- for example, if you give a medication, the null hypothesis is that the medication is no different than the -- the placebo, for example.
Q. All right. And in that case, the -- the medication would be the independent variable and the -- and the health endpoint that you're looking at would be the dependent variable, correct?
A. That's my understanding.
Q. All right. And do you know how multiple regression analysis looks at the relationship between the dependent and independent variable in order to examine the issue of the presence of bias?
A. As I already stated, I would hire a statistician to make sure that my analyses were correct.
Q. Okay. Dr. Speer, someone from reading that

answer might not understand whether or not you have knowledge in that area or not.

A. As I've already stated, I have limited knowledge in that area, but I do know how to get help.

Q. Okay. Do you know -- do you know, for instance, how -- what a regression line is?

A. Only in the broadest general concepts.

Q. All right. And what is your understanding in the broadest general concept of it?

A. That it's a regression line.

Q. All right. What does -- and what does that line represent?

A. I cannot tell you.

Q. Do you know what the product of regression analysis is?

A. I think I've answered this question a number of ways, but I'll try once more. I am not a statistician and I'm not an epidemiologist as trained. Therefore, I would refer you to a statistician or an epidemiologist to answer your question.

Q. Please do not think I'm trying to be impertinent. I'm trying to ask a different question. And I didn't make myself clear I

don't think. There is a statistic -- and it is a -- it is a statistic that is associated with the product of regression analysis, and it has a particular name.

And I'm just wondering if you know what the particular name of that statistic is that is produced as a result of a regression analysis.

A. I probably do. But given how you phrased the question and not having a statistics book in front of me to refresh my memory, I cannot tell you.

Q. All right. Do you know what "R" is?

A. Oh, that indeed -- Pearson's R?

Q. Yes.

A. Okay. I have studied Pearson's R back in 1962, I believe, in statistics.

Q. All right. And what does "R" tell us?

A. "R" is a relationship between various factors. The higher the "R," the more the relationship.

Q. It's correlation?

A. Correct.

Q. All right. And what is -- how does correlation relate to a regression statistic

if it does?
A. It probably -- I believe it does. And I can't answer your question.
Q. Okay. Do you know what types of regression analysis are appropriate? Well, let me go back one.
Are different types of regression analysis necessary depending upon the type of dependent variable that is used?
A. Indubitably.
Q. All right. And do you know what the different types of regression analysis are?
A. No. That's why I hire statisticians.
Q. All right. So in terms of whether or not linear regression analysis or logistic regression is appropriate, that -- that would be a question you're not prepared to answer?
A. Correct. I'm designing the study. I would -- I always -- if you're dealing with numbers and variables, I involve a statistician up front to help design the study.
Q. All right. Would the same be true, for instance, about when a stratified analysis is appropriate, that that's something that would

be beyond your realm --
A. Correct.
Q. -- you'd hire somebody else to look into that?
A. If it's multiple stratifications, yes.
Q. Is it your understanding, Dr. Speer, that a "P" value in a study measures only the extent to which a type one or Alpha error has been excluded?
A. No.
Q. What is your understanding then about what a "P" value is?
A. A "P" value in a given analysis tells you particularly to the analysis what are the odds that the findings are different from the null hypothesis. A "P" value of .01 says there is a one in a hundred chance that, given the numbers that you used in that study, there is a chance of one in a hundred that your results are wrong. However, "P" values depend a great deal on the power of the study, which depends on the number of patients and the numbers of observations made. You cannot really set up a study and use any statistical analysis that results in

a "P" value unless you've also taken into consideration the power of the study.

Q. All right. Do you know what a type one or an Alpha error is?

A. At one point in time I did.

Q. How about a type two or Beta error?

A. That's the opposite of the type one error.

Q. Okay. Which would be?

A. One says that you have made a conclusion that is erroneous in the favor of saying that there is a true difference in the results. And the other is the opposite of that.

Q. False negative versus false positive?

A. Correct.

Q. The -- in terms of the power of the study, is both the -- the level set for the type one error and the level set for the type two error, are they outcome determinative in terms of the -- of the study power that you're able to generate?

A. The higher the power, the better the data.

Q. What I was asking for was your understanding of how study power is or is not a function of the level set for type one or type two errors.

A. There is an equation to come up with power that involves both the Alpha and the Beta.
Q. All right. And is it your understanding that an examiner can set whatever values they want to in terms of their statistical analysis of data in terms of the P one or -- excuse me -- the type one and the type two error that they plug into that equation?
A. "P" is usually defined as not less than .05 in power. And the power is usually around 80 percent. All right. But, yes, you can put any numbers you want into the equation.
Q. In what -- what number do you generally accept as sufficiently ruling out a random association being found in the study?
A. As I implied, there is no such thing as ruling out. You're merely dealing with probabilities.
Q. Okay. The way -- is it your understanding that the way these statistical methods work, that no matter what values that you put in there, there is always the likelihood of a random association being demonstrated between the dependent and the independent variable?
A. I think you used the word "likelihood." I

think that's probably erroneous. Depending on the power of the study and depending on the analysis and depending on the "P" value, you can have results that are highly unlikely to be abnormal or in error of results, or you can have a "P" value that says that one time in 20 you'll have a possibility.

Q. All right. That would correspond with the "P" .05, would it not?

A. Correct.

Q. So a "P" value of .05 means that even if there was no true association between A and B, that you would reject the null hypothesis 1 out of 20 times simply on the basis of the random variation of the data and not because of any true relationship that exists?

A. Correct. In a single study that is correct.

Q. And what is the -- what is the "P" value that you view as the gold standard in terms of the -- the level at which clinicians like yourself believe that random variation has been sufficiently ruled out to consider that there's a statistically significant result that's been demonstrated in the study?

A. I think you just mixed apples, oranges and

pomegranates in there, but I'll attempt to answer the question.

Q. Well, no, then don't because I don't want you to -- to try and answer a question that -- that you think was confusing or that --

A. Okay.

Q. There is -- in your vernacular, do you consider "P" a level of the statistical significance of the data?

A. As I mentioned earlier, the lowest, if you will, "P" value that most individuals will accept as showing some degree of validity is .05.

Q. All right. Are you among that group?

A. Yes, but that doesn't mean the data is incontrovertible. Did I pronounce that word correctly? I don't think so.

Q. It sounded right to me.

A. Okay.

Q. Well -- and -- and you raise a good point. Is it fair to say that a statistical association is not of itself a statement regarding whether or not the independent variable caused the change in the dependent variable?

A. Depends. You've made an awfully sweeping statement. And I don't think -- I always operated under the rubric of never is -- there are never nevers and never alwayses. So the risk of the sun rising in the west is fairly small but it may occur given cataclysmic occurrences within the universe.
Q. Is your point there that statistics are merely measures of the probability of an association, or the statistical manipulations that are performed in epidemiologic studies are merely a measure of the probability of an association?
A. Statistics measure probability.
Q. All right. And there's a statement in the 1964 Surgeon General's document, for instance, that says "Statistical methods alone cannot establish proof of a causal relationship in association." Do you agree with that statement?
A. I'd like to see the entirety of the Surgeon General's report before I comment because I don't know in what context that statement is.
Q. Is it your opinion that statistical measure -- methods alone can establish proof

of a causal relationship?
A. They are the best method that I'm familiar with to establish relative cause, yes.
Q. All right. You had, as I looked over your resume, some substantial involvement in infectious diseases, did you not?
A. Yes.
Q. Were you a CDC?
A. No. It would have been fun.
Q. Did you, as a result of your work in infectious diseases, become familiar with Koch's postulates or the -- Koch's postulates?
A. Koch postulates are taught to you in medical school and in actually college.
Q. And -- and were those criteria which were developed historically to measure the presence or existence of a causal association between an environmental exposure and a disease outcome?
A. I can't remember the exact circumstances where Koch came up with his postulates. So I can't really answer your question.
Q. All right. Well, apart from the specific circumstances in which he developed his

postulates, was it your understanding when you learned Koch's postulates in medical school that they were criteria that were developed in order to assess the likelihood that an environmental exposure was causal or productive of a particular disease?

A. I don't know whether he was dealing with environmental exposures. That was the reason I answered the way I did just a bit ago.

Q. Okay.

A. Now, if -- and -- you know, Koch's postulates basically say that you do something to an animal or a human and a result occurs. You then take the something away and the animal either gets better or stays the same. You reintroduce the stimulus and you get the same responses you got the first time.

Whether that -- he dealt with environmental issues or more likely he dealt with some compound that he gave them -- gave the animal, I can't remember.

Q. Okay. And I -- I guess I unnecessarily confused you in the context of that question. I used the phrase "environmental exposure." And I was just struggling for a term to

describe some substance that was capable, potentially capable, of producing a disease.

With that qualification, is that a fair assessment of what Koch's postulates address?

A. As I've just told you that -- what I told you is my memory of what Koch's postulates are. If you'll get me a text, I'll be more than happy to review what Koch's postulates precisely are.

Q. All right. The -- have you studied the literature that has dealt with the issue of how one goes about making a causal determination on the basis of epidemiologic and other data?

A. I think I answered that a while back. I'm not an epidemiologist nor a statistician. Therefore, I would have had no cause to study methodologic literature.

Q. All right. Well, there is -- and you pointed out quite eloquently at the beginning of the deposition the shortcomings of case reports. Would it be fair to say that -- that at best case reports may generate a hypothesis about a potential association between exposure to something and a health endpoint but they do

not, by any means or measure, test that hypothesis?
A. In general, that's a fair statement.
Q. All right. And -- and that epidemiologic studies which, by the definition that we've just arrived at, do not include case reports are -- are one of the means by which we examine the hypothesis that there is an association between exposure to something and a disease?
A. Can you simplify that question a bit?
Q. I would -- if it's possible. I mean, I -- the question, I hope, has a lot of communicative impact, but --
MR. MINTON: Could you read it back, and I'll give it my best shot?
(Requested portion of testimony was read back)
Q. (By Mr. Minton) I'll start over.
A. Okay.
Q. Are epidemiologic studies a means by which we examine the hypothesis that an exposure to something may be related to a disease endpoint?
A. That can be an epidemiologic study.

Q. And that is indeed one of the reasons why epidemiologic studies are performed?
A. That is a reason.
Q. All right. There are other ways of examining a potential causal relationship between exposure to a substance and a health endpoint, are there not?
A. Yes.
Q. Toxicologists do that, don't they?
A. Probably.
Q. All right. Molecular biologists do that, don't they?
A. That's my understanding.
Q. All right. There are -- there are branches of science other than epidemiology that are actively involved in investigating potential causal associations, are there not?
A. I'm not even too sure you have to limit it to science. History examines potential causal relationships in trying to explain historical events such as wars and families. That in a way is an epidemiologic study. So I wouldn't necessarily limit what you're stating to science.
Q. Okay. Well, what I want to do is try and

limit it to health endpoints and -- and -- so what I'll do is re-ask the question.

And that is: In terms of investigating the relationship between exposure to a substance and a health endpoint, there are a variety of disciplines outside of epidemiology, medical disciplines, that look into those areas.

A. As a broad, universal statement, yes.

Q. All right. And -- and we've just covered that toxicologists, molecular biologists, perhaps even chemists, would look at those relationships as well?

A. If they -- if you're talking about randomized controlled trials, yes.

Q. And is there, to your knowledge, within the realm of epidemiology, principals or criteria that have emerged which medical practitioners and others use in terms of evaluating epidemiologic data to determine the likelihood that a -- an exposure to a particular substance may cause a particular health endpoint?

A. Given the universal nature of your question, there probably are.

Q. Well, are there -- what are the criteria that you use in terms of arriving at a judgment or opinion that exposure to a substance is causally associated with a health endpoint?
A. Would be the preponderance of evidence.
Q. All right. And what is the evidence about which you would look for a preponderance?
A. Depends on the question you're asking.
Q. All right. Well, let's -- let's --
A. If you're talking about, say, strychnine and you feed it to an animal and 100 percent of them die, then it's probably very highly likely that strychnine is a poison.
Q. Okay. Because that would then satisfy Koch's postulates, correct?
A. If you had a randomized controlled trial and you didn't know which substance you were feeding the animal. And then when you broke the code, all of the animals who died received strychnine, yes.
Q. All right.
A. Because Koch's postulates are really not statistical methodology.
Q. They look at whether or not a substance necessarily produces an effect, whether it's

sufficient to produce an effect, and whether or not you can replicate that effect by re-administration of a dose, and whether you can remove that effect by removal of the dose, correct?

A. In simplistic terms.

Q. Well, is that a fair analysis of how you view Koch's postulates?

A. In simplistic terms, yes.

Q. Is there some more sophisticated overlay that you think is appropriate to add to the enunciation of Koch's postulates?

A. Well, people really don't use Koch's postulates, to my knowledge, as a primary methodology to examine the question regarding null hypothesis. I mean, the gold standard is a randomized, double-blind controlled trial. Now, that is not always possible to do. So you move to the next stage of studies.

Q. The gold standard of determining a causal association is a randomized controlled clinical trial?

A. Blinded.

Q. Randomized controlled blinded clinical trial?

A. Randomized blinded -- double-blinded, actually -- now you've got me doing it -- yes.
Q. All right. If we don't have that then we go down the scale to what?
A. You can do epidemiologic studies. You can do observational studies. You can do non-blinded studies because sometimes it's impossible to blind. You try to randomize as best you can so that you -- and you like to have as many numbers as you can because that decreases the chance of bias and it decreases the confounding variable issue that you talked about earlier.
Q. And the reason that the randomized double-blinded controlled clinical trial is considered the gold standard is because in terms of experimental methodology, it controls best for those factors in terms of any study methodology that we're aware of presently today?
A. It basically tries to remove bias from the equation.
Q. All right. And as we -- as we move down the scale into epidemiologic studies and

observational studies, we don't have the control over those variables that we have in an experimental setting, correct?

A. You have natural selection.

Q. Meaning what?

A. Covered.

Q. Meaning that the -- that the experimenter has to -- has to deal with the -- the natural environment with which he is presented. He cannot create one experimentally?

A. Correct.

Q. All right. And because he has to deal with that environment with which he has presented, that environment has potential biases, confounders, that sorts of thing?

A. Potential.

Q. All right. And if one of the methodologies that is used below the gold standard -- strike that. Let's start over.

Are you familiar with the literature that deals with the issue of how one goes about making a judgment or opinion of causality based upon data that is contained in epidemiologic studies?

A. I think the answer is no.

Q. Thus, as you sit here today, you're not familiar with criteria enunciated by various authors that say, "Here are the things you should look for and examine in terms of making a judgment based on epidemiologic studies that there is a causal association between this exposure and this outcome"?
A. Having phrased your question somewhat differently, I may be.
Q. All right. And what is it that you may be familiar with that bears on the answer to that question?
A. Give me an example, and I'll be happy to respond.
Q. Well, what -- what are --
A. Because I'm not too sure where you're -- where you're going to or coming from, actually. I thought we were going to be dealing with issues of my opinions on products of tobacco on babies. And we seem to have spent a great deal of time on statistics and epidemiologic issues in which I've already told you I'm not an either -- an expert on either. But if you want to continue along this vein, it's your time.

Q. Okay.
MR. MINTON: Take a break?
THE COURT REPORTER: May I -- is it a good time to change my tape? I just don't want to interrupt you.
MR. MINTON: Sure. We'll come back and then we'll have --
THE VIDEOGRAPHER: The time is 10:40 a.m. We're going off the Record.
(A recess was taken)
THE VIDEOGRAPHER: The time is 10:45 a.m. We're on the Record.
Q. (By Mr. Minton) Doctor, before we broke, we were discussing the literature which has dealt with how one goes about the interpretation of data from epidemiologic studies to make a judgment about whether or not a particular exposure causes a particular health endpoint.
And what I'd like to ask you is: First of all, are you -- are you -- I think you've told us, but I just want to make sure. Are you unfamiliar with that body of literature?
A. It's not something I read on a routine basis.
Q. All right. In terms of the criteria that are

applied in the context of that judgment, that is something that you presently do not know?

A. Well, I'm -- you know, I may or may not be familiar with those criteria. I don't read that literature on a routine basis as I've already stated. And I don't know exactly what your definitions are. And if you would give me some of those, then I perhaps could respond a little more intelligently.

Q. My definitions of what?

A. Those criteria.

Q. Okay. Well, that's -- that has the cat-out-of-the-bag problem. But what I'm interested in is in knowing if -- if you know what any of those criteria are.

A. I may well. But, you know, as I've already stated, I don't read the literature. And although I may know something, unless you can say, "Is this white or black," I can't tell you whether it's white or black.

Q. Okay. Have you -- in the context of providing your opinions as expressed in Exhibit 1 and in Exhibit --

A. Try 6.

Q. -- 6, did you make some sort of methodologic

review of the literature?
A. No.
Q. Have you ever made a methodologic review of the literature in the context of any of the opinions that are contained in Exhibits 1 and 6?
A. No.
Q. Do you have an idea, for instance, of what percentage of articles that you have reviewed that deal with the issue of the health effects of maternal smoking and some adverse fetal outcome?
A. I'm sorry. Perhaps we could read that back.
Q. I can just rephrase it.
A. Okay.
Q. Do you have an idea of what percentage of the literature that you've read that deals with the issue of maternal smoking and adverse fetal outcome?
A. Now, are you asking what percentage of the literature states that there is an adverse --
Q. No.
A. -- effect or what percentage of the entire literature that I have read for 25 years deals with tobacco?

Q. The latter.
A. I have no earthly idea. It's probably a relatively small percentage.
Q. All right. And of -- of -- as it is a relatively small percentage of what you have read yourself, do you -- do you have any idea of -- in terms of what you have read regarding maternal smoking and adverse fetal outcome, what percentage of the overall body of literature that deals with that issue is that you have read?
In other words, you've read a slice of the pie that deals with maternal smoking and adverse fetal outcome. Can you give us some idea of how big that slice of the pie is in terms of that overall pie?
A. I'm not too sure I understand your question once again, and I apologize for being obtuse. I read about babies. I read about what diseases babies have. I read about how to hopefully fix them. I read about influences on how they get to where they are. The literature on tobacco and its effect on babies is a small piece of that. It is a piece. How big a piece, I can't tell you.

You know, it's there in the overall context of caring for babies.
Q. All right. Is it less than 5 percent of what you've read?
A. Probably.
Q. All right.
A. Because -- the reason is tobacco may cause prematurity, for example. And it does. Once the premature baby gets to me, I don't care particularly why he's premature. I have to deal with the prematurity of the baby. So the fact that mother did or didn't smoke is immaterial to the fact that I have to take care of the baby.
Q. Is that -- is that going to be true with respect to the gamut of opinions in Exhibits 1 and 6, that in terms of your clinical activity, the etiology of the condition that you're presented with, by the time you're presented with it, is essentially irrelevant to you? You're trying to make the baby better. You don't care what caused it. Is that --
A. Well --
Q. I'm just trying to understand where you were

coming from in your last response.
A. It depends on how you define "care." I mean, I care that things cause prematurity. I can't do anything about it when I get the baby who's premature.
Q. If I could take those words back and -- and scrub them off the page, I would do it. I didn't mean to imply that -- that you didn't care in that sense. What I -- what I so clumsily was struggling for -- and I'll try again.

By the time a patient has reached your office, would it be correct to say that your clinical mission is to try to improve that patient's condition? And in the context of tobacco, whether tobacco caused it or something else caused it, is not something that effects your clinical mission?
A. In most instances, correct.
Q. All right. There are instances that -- where that's different, though?
A. Not necessarily at that point in time instantaneously. But, for example, if I have a patient who after being hospitalized has chronic lung changes, I certainly do not wish

that patient to go back into a tobacco environment because that's going to make -- potentially make that patient's lung condition worse.

So I will then, at that point, be concerned as to whether or not the family smokes. So it depends on the continue of my care as to whether or not I have an active interest in whether the mother or father smoke or more of a passive interest.

Q. Chronic lung conditions would be -- and that's chronic lung conditions in the baby?

A. Correct.

Q. All right. That would be one instance in which you would have an interest in a smoking history?

A. Correct.

Q. All right. It --

A. And I have an interest in a smoking history when I get things. Because if I have a baby, for example, that is born of a mother with preeclampsia and two pack a day smoking history, I'm going to be prepared that that baby is going to have more problems as a neonate than if the mother just had mild

preeclampsia.
Q. Is -- is maternal smoking negatively or positively associated with hypertensive diseases, maternal hypertensive diseases?
A. It is -- I don't think it necessarily has an effect on preeclampsia, per se.
Q. Okay. By that, you mean maternal smoking does not?
A. Correct.
Q. All right. Then how would mother's smoking enter into the treatment equation for a baby born where the mother had preeclampsia?
A. Because maternal smoking appears to be an independent variable in regards to small size in babies. And because of the vaso -- it's thought that because of the vasoconstriction and material circulation leading to the uterus you decrease the amount of nutrients that the baby has. If the mother also is preeclamptic, that condition also does a similar...
Q. So in a -- in a mother who has hypertension or who is preeclamptic, you would -- you would caution them not to smoke?
A. If I were an obstetrician, I would strongly

caution them not to smoke, yes.
Q. All right. But you don't get into that because you --
A. I'm not an obstetrician.
Q. Okay. The -- would it be correct then to say that the one area where history is significant to your treatment is where there is some chronic lung disorder in a baby?
A. That was one example.
Q. Okay. Are there others?
A. Well --
Q. We discussed one, but then you told me you weren't an obstetrician so you didn't really get into that.
A. Right. When we take the maternal history, we always inquire whether substances are used; tobacco, cocaine, alcohol, medications that are prescribed, et cetera. It's just part of a history. We're supposed to do that. And then, depending on our findings in the baby, the history may or may not be relevant.
Q. And -- and we mentioned one situation where that history is going to be relevant or might be relevant. And that's where a baby is born with a chronic lung problem.

A. No. No. That's after the treatment of the prematurity and all other underlying conditions the baby results in having a chronic lung problem --
Q. Okay.
A. -- and is about to go home.
Q. What -- and I -- you know, I just can't seem to ask you the correct question here. I'm trying hard. There -- is there a situation in which the mother's statement about smoking history affects treatment outside of the example that you've given us regarding a chronic lung problem in the baby?
A. I think I just did. But an example of smoking plus preeclampsia.
Q. Okay. But then I understood you to say that was -- that was an academic issue with respect of you because you're not an obstetrician --
A. It's only an academic issue so far as my counselling the mother. It is a -- it is a very real issue if I'm now faced with a small for gestational age, undergrown infant, who is a premature.
Q. Okay. So --

A. Because it may have made his condition worse. One by causing prematurity; and two by causing undergrowth.
Q. And how is that going to affect your treatment protocol with respect to what caused it? Would it make any difference what caused the baby to be small for gestational age as long as those two conditions coalesce, preeclampsia and small for gestational age?
A. Well, if mother didn't smoke, perhaps the baby wouldn't be small and wouldn't be born as premature as he is.
Q. I understand that. But in terms of the treatment protocol that you apply -- would it be a fair statement to say that -- that each of the health endpoints that you've identified in your expert disclosure document are multifactorial, there are numerous causes of each?
A. Yes.
Q. And so -- you know, we've been discussing one. And -- and there are numerous causes and -- known causes and -- and unknown causes as well that apply to a baby being small for gestational age, correct?

A. Correct.
Q. All right. And does it make any difference from within those universe -- within that universe of causes or potential causes? If that mother presents with preeclampsia and has a small for gestational age baby, does -- do the precipitating causes matter to you in terms of the treatment protocol that you apply? Would the -- the reason why the baby was small for gestational age enter into some change in the treatment protocol?
A. As your question is phrased, no.
Q. Okay. So with respect to the treatment protocols that you are called upon to apply as a clinician, the -- the one area in which that treatment protocol may be different given a -- a mother's smoking history is where there has -- where there is chronic lung dysfunction in the baby?
A. What you -- what you -- what you've done is you've taken treatment protocols, the beginning of life, and attached smoking history of the mother at the point in time of discharge. And I'm not too sure you can do that, but you have. Maybe I better ask for a

restatement.
Q. Okay. Let me -- let me try it a little bit clearer.
MR. BLEVINS: Again, we're at 11:00. Maybe by the time we get back from lunch we'll have that issue revolved. Doctor, we will be back here at, say, 12:50, 12:45?
THE WITNESS: I probably will be back -- be able to be back by that time.
MR. MINTON: Well, do you want to make it 1:00 just to make sure that we're --
THE WITNESS: Well, why don't we try for 12:50.
MR. BLEVINS: And we'll just be here when you get here. How about that?
THE WITNESS: Yeah. That's fine.
THE VIDEOGRAPHER: The time is 11:00 a.m. We're going off the Record.
(Lunch recess was taken)
THE VIDEOGRAPHER: The time is 1:17 p.m. We're on the Record.
Q. (By Mr. Minton) Dr. Speer, could you tell us basically today what your clinical practice consists of?

A. I see patients, depending on the time of year and the schedule, at Methodist Hospital, Ben Taub Hospital, Women's Hospital, St. Luke's Hospital and Texas Children's Hospital, and our newborn infants who require some degree of medical assistance.
Q. All right. And is your practice confined to neonatology at this point?
A. Correct.
Q. All right. And you're on staff at all four of those hospitals -- or was it five of those --
A. Five.
Q. -- of those hospitals?
And do you -- do you see and treat persons from the Texas Medicaid population?
A. Oh, yes.
Q. Do you know what a person's pay status is when you treat them?
A. No.
Q. So the -- the answer that you see and treat patients from the Texas Medicaid population is based on your belief and awareness that you're treating people from all spheres of life, and that Texas Medicaid patients would

be among those people and not because of any specific knowledge you may have had regarding the pay status of particular individuals?

A. In certain instances I may have retrospective knowledge of the pay status.

Q. All right. But you -- but you don't generally as you're treating a person know what their pay status is?

A. No.

Q. All right. Have you been provided any information by the State of Texas at all in connection with this lawsuit regarding the demographics of the Texas Medicaid population?

A. No.

Q. Do you know whether or not you see and treat patients who are part of the -- the Texas Employees' Health Plan?

A. It's possible.

Q. All right. But -- it's possible maybe not; it's possible maybe it is. You just don't know?

A. Correct.

Q. All right. Have you collected any data independently regarding the Texas Medicaid

population?
A. In what regard?
Q. Any demographic data to begin with.
A. We collect, as a section, demographic data on all of our patients into a database. So in that regard, being as to how about 40 percent of our patient population at Texas Children's is Medicaid, we have data on Medicaid patients. But we haven't specifically gone out and said, "We are going to collect data on Medicaid patients."
Q. Okay. Have you familiarized yourself with, for instance, the -- some mean or immediate income level of Texas Medicaid patients that you've seen?
A. No.
Q. All right. There isn't any range that you could give us for that statistic, is there?
A. Correct.
Q. All right. And would it be fair to say, Doctor, you wouldn't expect that statistic to be static from Medicaid population to Medicaid population, would you?
A. What do you mean?
Q. That the income level of a -- of a population

is not going to be a -- an immutable characteristic but, in fact, will -- will probably change from population to population?

A. What populations are you speaking of?

Q. Let's say we compared the Texas Medicaid population with the Florida Medicaid population. Would we have any scientific means of -- of knowing, to your knowledge, whether the immediate income level of those two populations is the same?

A. It's possible to get that data if you want it.

Q. All right. But as you sit here today, you have no knowledge how the income level of the Texas Medicaid population may compare with any other Medicaid population in any other state in the country?

A. Correct.

Q. Would the same be true of -- well, I'll just -- rather than confusing you with a general question, I'll try and be more specific. Do you have any data on what the -- the central tendency of the marital status of women that you see whose care is

paid by Texas Medicaid?
A. No.
Q. All right. So, you know, the percentage of those women who are married or unmarried is not something that's known to you?
A. Correct.
Q. All right. Is their age something that is known to you?
A. I know the age of the mothers who I take care of. But as a general rule, I do not know the age of the Medicaid population mothers.
Q. All right. How about their housing conditions? Do you have any data which to you characterizes the Texas Medicaid mother population on the basis of their housing conditions?
A. I have no demographic data in that regard.
Q. All right. How about the smoking habits of mothers -- and, by the way, just so that I don't have to -- to do this question in triplicate each time, when I specify the Texas Medicaid population, will you also include within your answers the Texas Employees' Health Plan and the charity care patients that you see? Am I making myself

clear? Probably not. I'll just back up.
A. We'll go along and we'll see.
Q. Okay. If there are -- if there are salient differences, would you be kind enough to point those out to us?
A. Depending on your question.
Q. Okay. The -- do you treat charity care patients as well?
A. We treat all comers.
Q. All right. And -- and would that include then persons whose care is not -- persons who can't pay for their own care who don't have any means of public or private insurance or Medicare or Medicaid and for whom the hospital simply absorbs the costs?
A. Or they're cared for in the hospital district, which is the same thing.
Q. All right. From within any of those populations, do you have data which permits you to estimate the smoking prevalence of pregnant women in those populations?
A. I could probably get it. I don't have that data.
Q. All right. And where would you go to look?
A. Oh, I'd probably go to the various

demographic databases that exist and do an inquiry. Probably I'd start with the Texas Department of Health.

Q. All right. And do you know whether they stratify their data on the basis of Medicaid status and smoking?

A. I don't know. Have to ask them.

Q. Okay. Do you have any knowledge of -- among any groups of Texas Medicaid recipients, charity care patients, Texas Employees' Insurance patients, if they smoke, how much they tend to smoke if they're mothers? I -- you know, I knew it. I just messed that question up right at the end, didn't I? If -- I'll start over.

A. Now, I know why we're going to have an extra day.

Q. It's my clumsiness for which I apologize. Some of these questions tend to get rather technical and they need to be phrased appropriately, and I hope you'll indulge me.

Among any population of mothers who smoke in the State of Texas, do you have any data which permits you to estimate how much those mothers smoke?

A. I don't personally have such data.
Q. Have you seen -- have you seen any?
A. I haven't looked for it.
Q. All right. And so would the answer to that question be no?
A. As I said, I haven't seen the data.
Q. Okay. Would it be fair to say then any changes in the demographics that we just mentioned over time is also something that's unknown to you?
A. I think if you take the population as a whole, the incidence of smoking has slowly fallen over the last decade, decade and a half.
Q. All right. And have you seen data that indicates that that general proposition is -- is true also for the Medicaid mother population?
A. It's less true for the lower socioeconomic group as opposed to college graduates.
Q. All right. And have you seen data which indicates how much less true it is?
A. I have seen data a number of years ago comparing those groups, but I haven't -- don't have it at hand nor have I seen

anything recently.
Q. All right. But as you sit here today, for instance, you wouldn't be in a position to reliably estimate the prevalence of maternal smoking in the Texas Medicaid population?
A. Correct.
Q. Do you have any data on the pregnancy outcome statistics -- and specifically adverse pregnancy outcomes -- that you are going to testify about with respect to the Texas Medicaid population?
A. Not at this time.
Q. All right. No one's given it to you and you haven't looked for it, correct?
A. Correct.
Q. All right. The -- the same would be true, I take it, for the Texas Employee Health Plan and charity care patients?
A. Correct.
Q. So, for instance, what the incidence of low birth weight outcomes is among Texas Medicaid mothers, you don't know?
A. No. I could find that out.
Q. All right. Where would you go to find that out?

A. That data I have seen. And I think it comes, again, TDH.
Q. But in -- in connection with preparing to give your deposition today, no one asked you to review that data and you have not reviewed that data?
A. Correct.
Q. All right. Is it fair to say that you're as prepared today to give your opinions in this case as you intend to be?
A. Not necessarily. It depends on what people wish me to speak on at the time of trial, I presume. And if there's new information that comes to hand or that is relevant, then I will address that new information.
Q. As of today, have you completed all the tasks that you've been requested to do?
A. I was not requested to do any tasks except for to serve as an expert in the field of neonatology regarding tobacco in mothers and babies. If there's someone, for example yourself, that wishes me to have a task, I will certainly consider the task.
Q. All right. But -- well, one of the -- one of the tasks you completed was a document

providing us with your opinions in the case, correct?

A. Correct.

Q. And in terms of doing that, providing us with a statement of your opinions in the case, you've done all the work that you intended to do, correct?

A. Up until this point in time, yes.

Q. All right. And as you sit here today, is there anything out there that you know that you intend to do that you would have done but for conflicts or -- or, you know, an unavailability of resources or time?

A. Well, I would have liked to have been able to read some of the depositions with a little more scrutiny and care. But as you pointed out quite aptly, time is not necessarily always present.

Q. I mentioned low birth weight babies. And -- and why don't we all get an understanding in terms of phraseology so that we can communicate on the most effective basis.

In terms of characterizing your opinions, would it be more appropriate to use the term "small for gestational age" rather than "low

birth weight" if we were going to compare that health endpoint with prematurity?
A. They're different.
Q. All right. And how do you characterize the difference between low birth weight in terms of -- of a growth restricted baby versus small for gestational age?
A. A low birth weight by the World Health Organization definition is a baby under 2500 grams.
Q. All right.
A. Small for gestational age baby is any baby at a given gestational age whose weight is less than the tenth percentile.
Q. All right. And in terms of a term baby -- and, again, so that we can communicate, what do you characterize as term?
A. A baby who has completed 37 weeks gestation. In other words, 38 weeks to 42 weeks.
Q. More than 37 weeks or 37 --
A. Has completed the 37th week and is now 38 weeks.
Q. All right. And in a baby who is exactly 38 weeks --
A. He's term.

Q. -- who weighs 2500 grams, where on the scale of -- if we had a histogram of birth weights, where would that baby fall in terms of percentile or decile?
A. Well, I'd have to get my curves out to be precise, but probably about the 15th or 20th percentile.
Q. All right. So at 3700 grams -- at -- start over.
At 38 weeks or more, a 2500-GRAM baby is somewhere in the neighborhood of the 15th to 20th percentile?
A. Maybe. I'd have to get the curves.
Q. All right. Well, I'm -- I'm trying to get your best estimate as we sit here. And I realize you don't have a textbook in front of you.
A. Well, but -- in order to give an answer to your question, I almost have to have a curve in front of me to give you an accurate answer.
Q. All right. Are there babies who at 38 weeks, though they weigh 2500 grams or less, are small but normal?
A. Yes.

Q. All right. And in any -- is that a function of the fact that -- that births in the United States tend to follow a Gaussian or normal distribution?
A. Correct as a general population statement.
Q. All right. So even without some pathology or pathophysiologic change, we expect, simply by operation of the natural distribution of birth weights, a certain number of perfectly normal 2500 gram or less babies, correct?
A. As a statement, correct.
Q. All right. And -- and within the approximately 15 to 20 percent -- but you said you wanted to check that number -- some of those babies are going to be small but normal. And others are going to be growth restricted, correct?
A. And some are going to be small and abnormal.
Q. Small and abnormal in some way other than growth restriction?
A. Correct.
Q. All right. And these -- again, just so I made my question clear, these are all term babies?
A. That's how you defined it.

Q. All right. In terms of a baby who is less than 2500 -- who is term, less than 2500 grams, and small for gestational age, what are the diagnostic characteristics of that baby?
A. Multiple.
Q. All right. With that qualifier, would you tell us what they are?
A. You're talking about a host of diagnoses. I will give you some. And it's not going to be an exhaustive list because I'd have to go probably run a database to make sure I was complete.

You can have babies who are term under 2500 grams who have trisomy 18, trisomy 13, trisomy 21, trisomy 8, trisomy 1, trisomy 2, trisomy 4, Cornelia de Lange's, multiple different types of dwarfism, rubella babies, CMV babies, babies whose mothers are small, babies who have osteogenesis imperfecta, babies who come from mothers who smoke as the only reason babies born of mothers with pre -- severe preeclampsia and intrauterine growth retardation. And there are undoubtedly a host of others.

Q. All right. The trisomy --
A. More trisomies too and duplications and deletions.
Q. Those are all chromosomal --
A. 4 P minus is another one.
Q. Are those all chromosomal abnormalities?
A. Uh-huh.
Q. All right. Now, within small for gestational age, are there both symmetrical and asymmetrical growth restrictions?
A. Correct.
Q. All right. And have -- have you studied the literature with respect to maternal smoking and small for gestational age to enable you to render an opinion whether or not the babies born to mothers who smoke, that there has been an association made in the epidemiologic literature whether or not those babies are -- have symmetrical growth restriction or asymmetrical growth restriction?
A. Depends on the patient.
Q. And in what way would it depend on the patient?
A. Well, asymmetric growth retardation with

sparing of the fetal head occurs first. And if the origin of that growth restriction continues, then you get symmetrical growth restriction. In other words, the fetal head doesn't grow.

Q. Would -- would it be a fair statement to say that whether or not a growth restriction is symmetric or asymmetric is dependent upon the mechanism by which the growth restriction is produced?

A. As a general statement that's reasonable.

Q. All right. Well, in terms of our biological understanding of pathologic processes, isn't that the most reasonable statement to make?

A. What I'm saying is -- and for the example of a rubella baby, those babies are symmetrically small. In the case of other conditions such as intrauterine growth retardation secondary to placental insufficiency, you may start off with asymmetric growth restriction, but you may end up with symmetric growth restriction on the same baby.

Q. All right. How about with respect to the growth restriction that has been associated

with maternal smoking?
A. It can be either.
Q. And how could it be either?
A. Because you have a cumulative effect. A baby who may be born prematurely may show low birth weight but you've got a relatively small -- excuse me -- a relatively normal sized head. But if that baby under those same intrauterine conditions went longer, that baby may well have immunogen of head growth as well as body growth.
Q. All right. Have you studied the literature to determine if there are articles that have looked at the anthropometric measurements of babies who have mothers that smoked?
A. No.
Q. And so is it a hypothesis that you just stated to us that -- that you have not checked in the literature to determine whether or not there is a characteristic pattern of growth restriction seen in the -- the offspring of mothers who smoke?
A. I've seen both.
Q. Clinically you have seen both?
A. Yes.

Q. All right.
A. And not just on one occasion.
Q. But in terms of what the epidemiologic literature says, as you sit here today, you don't know?
A. Correct.
Q. All right. The other -- besides small normal, you mentioned small abnormal. To distinguish it from small for gestational age and -- well, let me back up. Perhaps I overspoke there.

I understood that you had broken out into three tiers: Babies who were 38 weeks or more weighed less than 2500 grams; there were the small for gestational age babies; there were the small normal babies and the small abnormal babies who were not SGA. Did I get that wrong?
A. That's all right.
Q. All right. Within that third tier, the small abnormal who aren't SGA, what are those babies?
A. Those are babies that subsequently are shown to have defects in their mental development. They may have cerebral palsy. They may have

mental retardation. They may have learning disabilities. They may have some -- they may have some of those conditions that I named earlier. And they are not just -- they're not SGA, but yet they have those conditions that I named earlier that you asked me for.

Q. Okay. I think I understand. There -- it's largely a group with chromosomal abnormalities but it may include others?

A. Actually, it may be what appear to be normal babies at the time of delivery and in the normal newborn nursery, or the newborn nursery, whether it be a special care nursery or otherwise, that subsequently are found to be abnormal.

Q. Do you have any estimate in any population of how those three tiers break up to comprise the under 2500-gram birth outcome?

A. I don't have any data at hand, no.

Q. All right. And so there is a percentage of idiopathic and normal under 2500-gram birth outcome that exists?

A. What do you mean by idiopathic?

Q. Not ascribable to any particular cause.

A. You're saying that there are outcomes that

are idiopathic?
Q. Well, perhaps I'm, you know, not using the correct language. We -- we've established that somewhere in the neighborhood of 15 to 20 percent of birth outcomes of term babies are below 2500 grams and -- and -- correct?
A. With the caveat that I haven't looked at the curve. So we may be over or under -- underestimating the numbers.
Q. All right. And that there are at least three compartments into which those --
A. Well, actually, there are only two.
Q. Small normal and small for gestational age?
A. No. Small normal and small abnormal. SGA is merely a subset of the small abnormal or sometimes a subset of the small normal.
Q. Very good. And -- and how those two categories divide in terms of comprising that group of the 15 to 20 percent of birth outcomes you can't say?
A. Term.
Q. Yes.
A. We're still talking about term babies.
Q. At term?
A. Correct.

Q. All right. Is there -- is there any way clinically of telling a -- a small normal baby from a SGA baby who has symmetric growth restriction?
A. It's easy to say who's SGA because that's a weight definition.
Q. All right.
A. So, yeah. It's very simple. Now, if you want to say whether he's asymmetrically SGA or symmetrically SGA, yes, again, you can because you look at the normal curves for weight, length and FOC.
Q. All right. What -- what would differentiate? If we had two babies, both of which or whom weighed 2400 grams, and they both -- and -- and one fit into the category of small normal and one was SGA with a normal growth restriction, what would be different between those two babies?
A. A normal growth restriction?
Q. Symmetrical growth restriction.
A. Then let's back up to get the whole question then.
Q. Okay. Let me ask it again. If we had two babies here, one was 2400 grams and weigh --

and was characterizable as small normal; the other was 2400 grams and was small for gestational age but with a symmetrical growth restriction, what would be different between those two babies clinically?

A. Well, 2400 grams, I don't know if you're SGA. You may be pretty close to it, but I don't know if you're truly SGA.

Q. All right. What -- what is the clinical cutoff for SGA?

A. 10 per -- weight less than 10 percentile for the gestational age of the patient.

Q. Will that vary significantly from population to population?

A. You will have more small babies and thus more SGA babies in certain racial groups than others.

Q. All right. Is the -- would it be correct to say that the -- the birth weight distribution for black or African-American babies is shifted about one standard deviation to the left of the birth weight distribution for white or Caucasian babies?

A. A whole standard deviation for what, the whole population --

Q. Yes.
A. -- or compared to the white population or what?
Q. Comparing blacks to whites.
A. Okay. So you're saying -- your question is, is the mean birth weight for the black population a full standard deviation lower than the mean birth weight for the white population?
Q. Correct.
A. I don't know if it's a full standard deviation. It's lower, but I can't tell you how lower.

MR. MINTON: Would you mark that as the next exhibit, please?

(Speer Exhibit No. 11 marked for identification)

Q. (By Mr. Minton) The first thing that will be evident, Dr. Speer, is that I'm no artist, from looking at Exhibit 11. But what I tried to do crudely on Exhibit 11 is to plot weight against number of outcomes in order to give a histogram of -- of birth weight outcomes. And the "W" would be the mean in the white population, and the "B" would be the mean in

the black population.
And before we attempt to put any numbers or indices on there, does the shift left for the black curve comport with your understanding of the true nature of the data?
A. There is a shift to the left of the black curve. Whether it is great as illustrated by your drawing, I can't tell you.
Q. All right. And whether it is in the neighborhood of one standard deviation is something that -- that you don't know at this point as well?
A. Correct.
Q. All right. Do you know, for instance, the difference in decile or percentile where 2500 grams would fall for the white versus the black population?
A. No. I'd have to go back to the data and find out what -- where on the curves it fits.
Q. All right. In terms of accurately characterizing small for gestational age, then, we would have to take racial differences into account?
A. If you use a purely racially-based curve, then the SGA child, by definition less than

the 10th percentile for the body of patients examined, the birth weight on the black patient who is described as SGA would be lower than the birth weight of the white patient described as SGA.

Q. And therefore --

A. Except we don't split the curves.

Q. When you say "we don't split the curves," who are you referring to?

A. We, neonatologists, use the curves that are provided to us. They are not based on black, white, Latin-American or other racial types.

Q. All right. Is it -- is it fair to say that there is, in general, twice the number of low birth weight babies if -- if -- if a standard cutoff is used for both blacks and whites, that there is about twice the incidence of low birth weight among black mothers as there is among white mothers?

A. I don't know whether twice is the correct number. I know there's an increased number of low birth weight babies in the black population compared to white.

Q. All right. Why is that?

A. Good question.

Q. We don't know, do we?
A. No, we don't.
Q. All right. If a -- never mind. Strike that.
Would it be fair to say that since we don't know what the percentage of babies under 2500 grams are that are small but normal as opposed to SGA, we likewise do not know what percentage of babies under 2500 grams in any particular population are going to require a level of care different than babies born above 2500 grams?
THE WITNESS: Would you please re-read that?
(Requested portion of testimony was read back)
A. Well, we do know -- I can't quote you the numbers, but we do know the percentage of babies who are under 2500 grams in term and the relative percent that will be expected to be normal versus abnormal. We know that babies who are under 2500 grams as a general rule will require more medical services than those babies above 2500 grams, because the below 2500 grams includes prematures. So I'm not too sure I quite understand your

question; but, hopefully, I've been responsive.

Q. (By Mr. Minton) You mentioned that we do know the relative percentage of babies who are small but normal and SGA in the under 2500-GRAM classification.

A. Well, as I've already said, SGA goes from the youngest survivor, which is 23 or 24 weeks, up to term. I mean, there's going to be SGA babies in each one of those categories. We know the relative survival of a baby who is born SGA versus a similar baby at that same gestational age that's AGA. We know that the SGA will die more frequently and has more medical problems at that given gestational age. We also know, as a general population, what the outcomes of babies under 2500 grams are, and we can subdivide that into categories either based on gestational age or weight and their given outcomes.

Q. The -- the problem I guess I put in my question was that I didn't specify term babies. And if we have -- as I understood your testimony, and I maybe got it wrong, there are babies who are term but under

2500 grams.
A. Right.
Q. There are -- there are babies who are small but normal, and there are babies who are small for gestational age.
A. And there are small but abnormal with the small for gestational age being a component of both the normal and the abnormal population.
Q. All right. And we don't have a statistic that -- that breaks out for us how each of those -- what percentage of the total under 2500 grams of term babies each of those comprises, correct?
A. Yeah, I think you probably do.
Q. What is it?
A. I don't have it, but I'm pretty sure it's out there.
Q. All right. The -- the babies who are term and simply small but normal generally are not going to require medical care that is different from a baby who is over 2500 grams and normal, correct?
A. No. Incorrect.
Q. All right. A baby who is term, 2500 grams,

and simply small normal is going to tend to require additional medical care?
A. As compared to a baby who is larger, correct.
Q. All right. And what -- what types of medical -- and I take it that's a rule or that is -- that is a generality which may or may not be true in individual circumstances.
A. Correct.
Q. All right. So the only way we're going to know whether a particular baby who's term, less than 2500 grams, but normal -- in other words, a small normal baby who is term at less than 2500 grams, the only way we're going to know whether or not that baby is going to require some additional medical expenditures is to have the information regarding that particular baby?
A. In a single individual case, yes.
Q. All right. And there are, then, a spectrum of possibilities for that baby which range from that baby is not going to need any care different from a 3200-gram baby to that baby is going to need care that is -- that is different from, in additional to, the care that a 3200-gram baby might receive?

A. Correct.
Q. All right. And for a baby who is small but normal, what are the additional treatments that may come into play that that baby may need?
A. First of all, it's diffi -- it's impossible to say at time of delivery that somebody is normal or abnormal. But if they are subsequently shown to be normal, the two most common problems of the small but ultimately normal baby who is term will have are temperature control, temporary maintenance, and hypoglycemia or glucose homeostasis. They may also have some problems with calcium balance. Those are the three most common findings in the ultimately proven to be normal but small baby at term.
Q. Temperature maintenance, hyperglycemia, and what was the third?
A. Hypoglycemia.
Q. Hypoglycemia.
A. Actually, just call it glucose homeostasis, will serve. And the third one is calcium homeostasis.
Q. Okay. And temperature maintenance means

what?
A. The ability to keep yourself at normal body temperature.
Q. All right. And how is that clinically accomplished?
A. By the metabolic processes that you and I enjoy to maintain our energy output and thus keeping ourselves warm.
Q. All right.
A. We are mammals. We are homeotherms.
Q. Okay. That's how it's done in the human body. If it needs to be externally mediated, how is it done in a hospital?
A. With an incubator or a radiant warmer.
Q. All right.
A. Or sometimes turning up the thermostat in the nursery.
Q. All right. And glucose homeostasis and calcium homeostasis, those are accomplished through an I.V.?
A. If we cannot accomplish it by feeding a formula or breast milk, it's accomplished by an I.V.
Q. And are those the same treatment modalities that may or may not apply to a baby who is

small for gestational age?
A. Correct. Although the small for gestational age has a tendency to have more severe problems in all three areas.
Q. Are there any significant areas that have thus far been omitted to describe the treatment modalities -- the probable treatment modalities for an SGA baby that haven't been discussed for a small normal baby?
A. It depends on what causes the SGAness.
Q. All right. What would be the additional treatment modalities if -- if the cause of SGA differs?
A. It depends on the cause. I mean, the patient may need a ventilator; may need antibiotics; may need antivirals; may need, you know, various studies of the cardiovascular system; the head, the kidneys, the endrocrine system. It depends on what the cause of the SGAness is. SGAness frequently is associated with other things, not just being small.
Q. All right. So there's a wide variety of treatments that may apply to an SGA baby, depending upon that caused the SGA?

A. Right.
Q. All right. So we wouldn't want to look, for instance, at costs attendant to SGA babies overall in order to predict costs from a particular form of SGA because those two may be dramatically different?
A. It depends on the population size and the influence or lack of influence that having an SGA baby or babies as part of that population are. It may have utterly no bearing if you have a large enough population size. If you have a very small population with lots of SGA babies, it will have a major bearing.
Q. Well, there are particular types of treatment modalities that are seen in particular causes of SGA that aren't seen in other causes of SGA, correct?
A. Correct.
Q. And some -- some particular causes of SGA tend to create particularly high medical expenditures, correct?
A. Yes. And just the opposite is also true.
Q. There are particular types of SGA that produce characteristically low medical expenditures as well.

A. Correct.
Q. Would it be correct to say that the -- that the types of causes of SGA that are associated with higher medical expenditures include those where surgery is necessary, where respiratory support is necessary and where -- well, are those the two major compartments?
A. We're talking about all SGA babies?
Q. What I'm trying to do is to see if there are -- if there's a universe of quote/unquote "expensive items" in terms of SGA babies or there are universes of.
A. Depending on the gestational age of the baby and the cause of the SGA, there may be very expensive things done for that particular patient, and among which are surgery, potentially, respiratory support, potentially, hospitalization, potentially.
THE COURT REPORTER: Excuse me. I'm going to need to change my paper. Would it be a good time to go ahead and do that?
THE WITNESS: Now is a good time.
MR. MINTON: Sure.
THE WITNESS: He's cogitating and

I'm waiting.
Q. (By Mr. Minton) All right. Dr. Speer, do you have any knowledge regarding the reimbursement structure in the Texas Medicaid population related to specific neonatal conditions, in other words, what the reimbursement policies and procedures are, depending upon what -- what the clinical problem is?
A. They are based on CPT codes.
Q. All right. And the reimbursement policies are based on CPT codes?
A. Yes.
Q. All right. And do you know which -- what is a CPT code?
A. A CPT code is a coding structure developed in part both by -- my understanding is the AMA, other medical organizations and HICFA come up with a code book that's called the CPT code. And there are various codes for various procedures and length of stay or hospitalizations.
Q. All right. And does the CPT code translate an ICD9 diagnostic code with an amount that is reimbursable through Medicaid?

A. In some instances, it may; but in our instance, it doesn't.
Q. Well, then how does it -- how does it determine the amount that's reimbursed?
A. We have -- in the neonatal ICU, they are bundled to codes. They are bundled.
Q. Bundled to what?
A. To whether you have initial hospital day, ICU unstable or ICU stable. That's it in the ICU. If you go to the step-down units, then there are, I think, four or five codes that address the complexity of the daily hospital care. And there are also consultant codes that are used in Level 2 settings. But the Level 3 settings or the ICU settings are all bundled codes. They include procedures; they include medical support; they include the daily care delivered on a 24-hour basis for that patient.
Q. So the -- let me make sure I understand this. In an ICU -- and is this specific to Texas Methodist Hospital or --
A. It's a neonatal ICU. It can be Women's Hospital or it can be Texas Children's Hospital.

Q. All right.
A. It can't be Methodist because there is no neonatal ICU at Methodist.
Q. All right. But in that ICU, there are for the ICU three bundled codes; and one says it's uncomplicated, and one says it's complicated. And what was the third?
A. Stable, unstable and initial admission day. 99295 is the initial admission day into the intensive -- neonatal intensive care unit.
Q. All right. And how will knowing what that code is for a particular individual tell you how much is going to be reimbursed by Medicaid?
A. Because the code stays the same, whether you're a Medicaid patient or a nonMedicaid. We submit a bill. Medicaid gives whatever discounted fee that they pay, and we get paid.
Q. All right. So for a particular Medicaid reimbursement, if a -- if a -- if the code for patient "X" is two days of stable ICU, that will translate into a fixed dollar amount?
A. Correct.

Q. All right. And how about -- well, let me just make sure I have a general understanding of how deliveries are treated in this area of the country.

Is it fair to say that -- that babies are divided into either neonatal intensive care unit treatment or nursery treatment?

A. There presently is -- this will change in the fall because the new guidelines for perinatal care will come out, and they subdivide care slightly differently. But presently, newborn care is divided into Level 1, Level 2, and Level 3.

Q. In an ICU?

A. In any -- just -- I've stated it as it is. Newborn care is divided, Level 1, Level 2, Level 3. ICU care is Level 3. Normal newborn care is Level 1. And everything else is Level 2.

Q. Okay. Are there -- are there codes for Level 2 care?

A. Those are the daily charge codes. And they depend on the complexity of the evaluation and management services as defined by HICFA that are rendered in order to charge a given

code. If you up code, you get paid more, but then you come under scrutiny of various regulatory agencies.

Q. And are there codes for Level 1?

A. They are a normal newborn code, yes. And there's a code for circumcision. And there's a code, I think, for discussion with parents. But there is a normal daily newborn code, and there is a discharge code, discharge day code.

Q. Okay. But would it be correct to say that an infant who has the most severe congenital abnormalities which require all of the intervention that can be brought to bear by the -- the NICU and is therefore coded as unstable will receive the same coding as an infant who merely -- who is in an NICU, Level 3 care, who is, for instance, receiving calcium homeostasis through an I.V.?

A. No. That patient wouldn't be in an ICU, the second patient.

Q. All right. What is -- what is the -- the least drastic, if that's the right word, intervention in an ICU? What's the minimum care modality?

A. It depends -- it really depends on the ICU you're speaking of. Some ICUs, in fact, many across the country, lump their Level 2 and Level 3 patients together and call the entire physical area an NICU. We, on the other hand, split them out. So in our NICU, the minimum expected intervention would be a ventilator. Either they are expected to be going to require ventilatory assistance or they already are. And once that decision is made that they are not, then they go to a Level 2 unit.

Q. But as I understand it, those expenses are then going to be coded or the care for those infants are going to be coded as expenses that will be the same for those two infants regardless of what it actually costs the hospital to produce those services?

A. Define your patient.

Q. The two babies, one which is receiving -- one baby who has a severe congenital abnormality who is receiving the highest and most expensive forms of support available in the NICU versus the baby who is in a ventilator.

A. Both patients, depending on if you -- if you

stay -- well, no, they are going to be different, because one is going to -- if he's just on a ventilator, he may be very stable on the ventilator without a whole lot of other things being done to that particular patient, so that may be an ICU stable charge; whereas, the other patient will receive an ICU unstable charge.

Q. All right. But so long as they are within the same tier, the charge is the same, regardless of what it costs the hospital to produce that service?

A. If you're within the stable or within the unstable, the billing to the insurance entities, the payer, shall we stay, it will be the same insofar as the medical billing. The hospital billing is a separate issue. And so the hospital billing would be more in a patient who requires more support unless there's a capitated fee arrangement that's already been, you know, arranged between the payer and the hospital.

Q. All right. Does Medicaid have such a capitated fee arrangement?

A. They have a per diem fee. They will have a

capitated managed care fee because that's where the state wants to go.

Q. What is the Medicaid per diem fee?

A. I don't know.

Q. And is it per diem based upon --

A. The per diem is based upon the bed charges. And then there are ventilator charges and I.V. charges and monitor charges and medication charges, et cetera, whatever the hospital is providing for that patient.

Q. But these are outside of the -- the hospital's normal cost coding?

A. No. Those are the normal cost coding that the hospital does. The CPT codes that we've been talking about merely are physician reimbursement fees.

Q. Okay. So everything we've really discussed up until now is simply what a physician gets paid rather than what the hospital gets paid?

A. That's -- when we got into CPT codes, you're talking about physicians.

Q. Okay. In terms of how the hospital is reimbursed, are you aware of how that occurs through Medicaid?

A. Presently it's my understanding that it's on

a per diem basis --
Q. All right.
A. -- plus everything that's done.
Q. And with respect to newborns, what are the -- what are the different classifications that can occur within that per diem?
A. Well, it depends on the hospital. Once again, there's usually a normal newborn daily charge that the hospital invokes. And then if the patient is, say, in a Level 2 unit, the hospital will charge a Level 2 fee for the room space. And then whatever other items of equipment or medications that are -- or lab tests that are required to care for the patient, those are on top of the daily room charge.
Q. Do hospitals within the State of Texas negotiate their own per diem Medicaid fees with Medicaid, or is there a standard charge?
A. I don't think so. I think it's a standard charge. They might want to.
Q. Are there a wide variety of treatment modalities that might be found within NICUs within the State of Texas?
A. Yes, because some people call themselves

NICUs when they are really not ICUs by our definition. It's like calling a community hospital a medical center.

Q. Are there -- are there significant areas within the state where NICU coverage simply doesn't exist?

A. True.

Q. And what percentage of the state would that be?

A. Are you talking about immediate access or distant access? Because there's distant access to NICUs throughout the state, but the community in which the patient is born in may not have immediate, within that community, an ICU.

Q. Well, I see the distinction you're making, but I also see a problem with it in the sense that ultimate access would, since it's possible to travel, pretty much embrace potentially anything. I guess a better term is practical access.

A. I don't think there's anyplace in the state -- and we're similar to other states, except maybe the very small states in the Northeast, where practical access is absent.

Around here we can move patients either by fixed wing or helicopter or ground transport. And there are NICUs that are in Houston, Galveston, Beaumont, Dallas, Fort Worth, El Paso, wherever Texas Tech is, Lubbock, Amarillo. And then there are smaller intermediate, less, perhaps, intensive coronary care, but they are a little lower in the classification Level 3s, in places like Lufkin and others.

Q. How about --

A. They are usually within an hour or two maximum from a major ICU.

Q. How about within the Texas Medicaid population? Could you give us an estimate of what the -- the practical access to NICUs is for mothers in the Texas Medicaid population?

A. I would anticipate it's quite high.

Q. All right. Are you willing to put a range or a confidence interval around a number?

A. No.

Q. Who -- in this area, is it the neonatologist who admit babies to NICU?

A. It depends again on the hospital. Sometimes pediatricians will admit patients to the NICU

with or without a neonatal consult. I think so far as the neonatal coverage in the state, it's -- there are over 140, and they are pretty well spread out. So you can probably find a neonatologist to consult in virtually all ICUs.

Q. Is there, to your knowledge, any standard set of admissions orders regarding admission to an NICU throughout the state?

A. I don't quite understand what you're asking.

Q. Well, physicians make decisions on which babies to admit to an NICU, correct?

A. Correct.

Q. All right. And there are certain criteria that those physicians may or may not apply in making that decision to admit a baby to an NICU, correct?

A. Correct.

Q. Is there some uniform set or standard set of criteria that you're aware of that guide that decision within the State of Texas?

A. There are some guidelines that are national guidelines in the peri -- in the guidelines for perinatal care that outline those patients that require higher levels of care

than can be provided within the community setting.

Q. All right. Do you have a uniform set of admissions criteria that you use?

A. As I've already stated, those patients who are thought to require or who do require ventilatory assistance are admitted to -- are in ICU.

Q. And is that the -- is that a unitary qualifier, or are there others?

A. That's -- that's pretty well it.

Q. All right. Babies who need ventilatory assistance?

A. Or who are expected to need.

Q. All right.

A. And sometimes we admit patients to a Level 2, and they subsequently need a transfer to the NICU.

Q. All right. And is the ventilatory assistance that they need, is that driven by respiratory distress syndrome, or is it driven by something else?

A. A variety of causes.

Q. All right.

A. It can be respiratory distress syndrome. It

could be apnea. It could be others.
Q. All right. Are those the two major reasons?
A. All patients admitted to the ICU, probably respiratory distress syndrome is the number one diagnosis.
Q. All right. And apnea number two?
A. It would be in competition with congenital heart disease, meconium aspiration, persistent pulmonary hypertension, septic shock, trans -- well, not transischemia.
Q. And -- and would the one -- would the conditions that you just named comprise the vast majority of babies who need ventilatory assistance?
A. Correct.
Q. Is there any reliable rule in terms of the birth weight of a baby in determining whether or not the baby is going to need ventilatory assistance?
A. Our database would imply that if you happen to be a gestation of 30 weeks, probably 50 percent of those babies will require NICU. If you're less than 30 weeks, particularly if you're, say, taking the 28-week population, about 90 percent of those babies will need an

ICU.
Q. And 30 weeks is going to correspond with what, about 17, 1800 grams?
A. Thirty weeks will be around 12 to 1400.
Q. All right. So at 12 to 1400 grams, 50 percent of babies are going to need ventilatory assistance?
A. Approximately.
Q. All right.
A. Higher on the low side and lower on the high side.
Q. Sure. Do you have any estimate at 2,000 grams what percentage --
A. 2,000 grams is 50th percentile for 34 weeks. A fairly small number of patients at that gestational age will need an ICU.
Q. Less than 10 percent?
A. Yes.
Q. Would it be fair to say that really only very low birth weight babies, if we define that term as 1500 grams or less, stand at least a 50/50 chance of needing NICU treatment?
A. As an entire population of less than 1500 grams?
Q. Yeah.

A. Our own experience is somewhat skewed because we have a lot of babies that are born in our hospitals because they are referral hospitals that are less than 28 weeks, and so we have far more than 50 percent in our population who are less than 1500 grams that end up in the ICU.
Q. Because of that referral bias?
A. I would anticipate.
Q. Okay.
A. As a general statement, across the entire universe, that's probably a reasonable statement, a 50 percentile, 50 percent below 1500.
Q. All right.
A. But it may be slightly higher.
Q. Okay. Do you have any data on whether or not -- strike that.
Have you, in connection with any of your opinions in this case, looked at any form of cost data for NICU usage among any population of mothers in the State of Texas?
A. No.
Q. Is it fair to say that in all likelihood the expensive treatment for a baby who receives

neonatal intensive care is going to vary from case to case?
A. Certainly.
Q. All right. And how much variability there is is not something that you're prepared to say here today?
A. It depends on the question.
Q. Well, could you -- could you tell us within a 95 percent confidence interval, for instance, you know, what the expense of that treatment is going to be?
A. For a given gestational age?
Q. We could start with that, yeah.
A. Basically, if you're born at 25 weeks gestation, it's going to cost about -- and you're a survivor, it will cost you approximately a half a million dollars -- a quarter to a half a million dollars before you get out of the hospital --
Q. Okay.
A. -- in charges. Now, whether you get paid that much --
Q. How about a -- how about a 30-week-old baby who is -- by the way, a 25-week-old baby who survives is a -- is a very rare phenomenon,

is it not?
A. About 25 weeks, above 500 -- say above 750 grams, about 80 percent.
Q. Well, 80 percent survival rate. But in terms of the likelihood of that birth occurring, would that be in the neighborhood of one-hundredth of one percent in terms --
A. What you're getting at, is it a small percentage of the total deliveries?
Q. Yes.
A. Yes.
Q. It's a hugely small percentage, if that's not too much of an oxymoron for you.
A. Right. But if they are here, that's what they cost.
Q. And -- and this -- this center is going to see more of those than -- well, this area of Texas, including this medical center, is going to see more of those because of the referral bias that exists?
A. We will see more -- the mothers will be referred in here still with the baby in the uterus, yes, within the womb. Now, you may have -- you do have 25-week babies being born in rural Texas who never make it to the

Medical Center. And in that instance, their mortality rate is quite high, and thus they are very inexpensive to care for.

Q. Now, you mentioned that figure for 25 weeks, which I believe we established, at least in terms of birth incidences, would be a statistical rarity. How about a baby who is 30 weeks and who is among the 50 percent who requires NIC usage and not the 50 percent that doesn't require NIC usage?

A. A baby born at 30 weeks will probably require hospitalization a minimum of five and a maximum of ten weeks, in general. Not all of that hospitalization will be carried out in the NICU environment. In general, most babies who are 30 weeks gestation do well unless they have a congenital abnormality. And if you're taking the patient that requires NICU care, the length of time within the NICU will probably average a week to ten days and with attendant charges.

The remainder of that patient's hospital stay will probably be carried out at a Level 2 setting. And depending upon the institution that is caring for that infant

and their price structure, the average cost within the NICU per day, including physicians' fees, will probably be somewhere in the range of 2500 to $3,000 a day. Outside of the ICU, the fees are probably closer to 15 to $1800 a day. So add it up, and you will come up with an approximation. But that may be different in Texas Children's compared to Hermann compared to Women's --

Q. All right.

A. -- which is called marketing.

Q. Okay. So we wouldn't expect to see the same charge hospital to hospital?

A. Not necessarily.

Q. All right. You mentioned marketing. Is that also going to be different from institution to institution because of the facilities that are available at particular institutions?

A. It can play a role.

Q. All right. And will it also be different from institution to institution depending upon what -- what perceived treatment modes or philosophies are deemed preferable at a particular institution?

A. Basic treatment modes are the same. There

are idiosyncrasies between individual physicians that may either lower or increase costs --

Q. All right.

A. -- and length of stay.

Q. Would it be fair to say that in some institutions NICU is -- is more selectively used than at other institutions?

A. I'm not too sure exactly what you mean by selectively used. Perhaps you could give me an illustration.

Q. Well, have you seen any audit of NICU usage from hospital to hospital that compared comparable cases and yet there was a different decision made with respect to NICU usage?

A. I have seen some of that data.

Q. All right. And would it be fair so that say that there is data out there which indicates that the decision to admit to NICU will differ on the same presenting characteristics from institution to institution, depending upon factors unique to that institution?

A. As a general broad statement, it's probably reasonable.

Q. Is the -- is 25 weeks the lowest cutoff in this area for admission to an NICU?
A. 23 weeks.
Q. And is that standard in this area, to your knowledge?
A. Well, 23-week babies do not do very well when there is question in the mind of the neonatologist and/or obstetrician as to the accuracy of dates. There are 23-weekers that are resuscitated and are in ICUs. And that is at least the standard that I'm aware of in most of the hospitals in this community.
Q. All right. I guess what I'm really asking about are -- there are -- there are survival criteria that are established in order for a baby to admitted -- to be admitted to an NICU?
A. If by "survival criteria" you mean an expectation for survival, yes.
Q. Yes. And would that include gestational age criteria as well as weight criteria?
A. Correct.
Q. All right. And is 23 to 24 weeks, for lack of a better term, a decision zone for admission to an NICU?

A. That would be a decision zone insofar as resuscitation and delivery with subsequent admission to an NICU.
Q. A physician might rightfully decide that a baby who was 23 to 24 weeks old bore such a poor chance of survival to withhold resuscitation efforts?
A. Correct.
Q. All right. And that is the -- the 23 to 24 weeks is the zone we're talking about that decision being made in?
A. Correct.
Q. And 25 weeks is the -- the earliest gestational age where we're outside of that decision zone?
A. 24 weeks.
Q. 24 weeks is where that occurs automatically?
A. Most often, yes.
Q. All right. And what is the -- what is the decision zone for weight?
A. 500 grams.
Q. All right. And is a 700-gram baby still in that decision zone?
A. No.
Q. All right. Below 500 grams?

A. Correct.
Q. All right.
A. And there are exceptions there.
Q. But routinely, care is withheld from a baby who weighs less than 500 grams?
A. Again, it depends on the patient. If the patient is a 26-27-week 485-gram baby, that baby is resuscitated.
Q. All right. And I didn't mean to imply that there weren't exceptions. I'm just saying that as a general rule, less than 500 grams, care is going to be withheld from the baby?
A. As long as that caveat that I stated is acknowledged.
Q. Dr. Speer, you've identified for us in your disclosure statement a number of adverse pregnancy outcomes for a neonate. And there is at the end of the statement an additional statement that says something along the lines of "as well as other effects." Are the named effects those for which you have prepared yourself to provide opinions in this case?
A. You're talking about --
Q. There is --
A. -- this document?

Q. Yes, I am.
A. Or are you talking about this document?
Q. As I understood it, they were -- they were essentially the same. But really what I'm talking about is Exhibit 6, not Exhibit 1.
A. All right. And where would you like to -- where are you drawing my attention to?
Q. Well, there are -- in terms of adverse pregnancy outcomes, as I read that document, there are five that are specifically articulated. And those are spontaneous abortions, reduced birth weight, premature births, abruptio placenta and placental injury. But then there's a statement "as well as other effects."
And I guess the focus of my question at this moment is simply, are there other effects that you have prepared yourself to testify about today that you intend to give opinions on other than the --
MR. MINTON: Am I wrong about that?
MR. BLEVINS: I mean, we haven't covered the increase in mental retardation, which is under Maternal Tobacco Smoking and Infant Complications. We haven't -- you

didn't discuss the neural developmental disorders or increased infant mortality or SIDS.

Q. (By Mr. Minton) Okay. The -- well, do we round out the list, then, with the statement of those additional topics?

MR. BLEVINS: Again, I'm sorry. I guess I'm going to go back to where you did in Dallas. I don't want to make an objection. But, I mean, the report speaks for itself to that effect. And I'm concerned about --

MR. MINTON: I'll rephrase my question, then.

Q. (By Mr. Minton) There are -- there are specific adverse pregnancy outcomes that are mentioned in that document.

A. Correct.

Q. Are we -- may we safely assume that the -- that the adverse pregnancy outcomes that are specifically mentioned in that document are the ones that you intend to testify about and provide opinions about?

A. That are within the entire body of the document, correct.

Q. Yes. Okay. And would it be fair to say that if a statement of an adverse effect is not found in that document that you don't intend to testify about it?
A. Today.
Q. All right. Or provide us opinions today about that adverse health effect?
A. There may well be items that are slightly different in nomenclature that aren't listed in the document that are related to things that are listed in the document that could be testified today.
Q. All right. In terms of spontaneous abortions, how -- how is that term used in that document?
A. It's self-evident. Spontaneous abortion.
Q. And what do you mean by the use of that term?
A. Well, it's not an induced abortion. It is an abortion that occurs by the mother going into labor and delivering an infant who is not alive.
Q. All right. Reduced birth weight, is that low birth weight and small for gestational age?
A. No. It's reduced birth weight for a given gestation.

Q. Premature births, is that any birth before the completion of the 37th week --
A. Correct.
Q. -- of gestation?
Abruptio placenta -- and I've seen that spelled several different ways, and maybe you can clear this up for us. Is that -- is it the Latin diphthong that goes at the end there, or is it -- is it truly p-l-a-c-e-n-t-a?
A. It's placenta.
Q. All right.
A. Although the British would probably spell it somewhat different.
Q. And that is a -- a tearing away of the placenta from the uterine wall?
A. Correct.
Q. All right. Placental injury is also mentioned in there. Is there a placental injury that you intend to address in your -- in your testimony, other than a placental abruption?
A. Correct.
Q. There is?
A. Correct.

Q. And what is that?
A. You have premature aging of the placenta with the increased infarctions and increased fibrosis, increased scarring.
Q. All right. There's a mention made in Exhibit 6 that -- that you intend to provide some opinions about mechanisms. And are those exhaustive in terms of the areas of mechanisms that you intend to get into? And I see that there are four there -- or four core statements. The fetal effects of all chemicals found in tobacco are not completely known. Carbon monoxide binds preferentially to fetal red blood cells and preference to oxygen, resulting in accentuated hypoxemia. Nicotine acts as a potent vasoconstricting agent, compromising blood supply to the fetus via a decreased uterine blood flow, and that nicotine is found in higher concentrations in the fetus and thus has direct fetal effects.
Are those the areas of mechanism that you intend to get into?
A. Correct, unless you ask the right question and it triggers another memory.
Q. Okay. And then under "Infant Complications,"

there's a statement about "Recent studies which show a relative risk of 1.75 for mental retardation in children of mothers who smoke."

There's another statement about other studies that have shown behavioral problems in children of mothers who smoke, raising concern over neural developmental disorders. And there's a statement about multiple studies that have found evidence of increased infant mortality due to maternal tobacco smoking with a relative risk of 4.0 of SIDS in children of mothers who smoked.

A. You didn't quite read it as well as you did before, but it's there.

Q. That's in substance the areas of --

A. Well, I can also go in -- you know, as a pediatrician, we certainly know there's a relationship between smoking and asthma in the household and other allergies, increased upper respiratory -- or respiratory tract infections in infants and children whose parents smoke. But I was talking about more the baby/infant diad as opposed to the older child.

Q. All right. Do you -- do you practice as a pediatrician as well?
A. No. I'm a neonatologist.
Q. All right. Have you been a pediatrician?
A. Yes.
Q. All right. When -- when did you practice as a pediatrician?
A. So far as actually seeing the complete spectrum of pediatrics was between the years 1970 and 1972, when I was a guest of the United States Navy. However, I serve on committees within the hospital that deal with pediatric issues, so I have been somewhat retrained in some of those areas as of late, although I do not see the children of the pediatric age group.
Q. I asked you earlier, Dr. Speer, about whether or not you had any publications with respect to maternal smoking and maternal or fetal health. And I gather the answer to that was no. But I failed to -- to ask you whether you had given any speeches on that topic. Have you given any speeches on that topic?
A. I would have to go back to my CV. I know that I've done some talks a number of years

back on drug addiction, but I don't think I spoke on tobacco at that time.

Q. All right. And I believe we covered this, but I just want to make sure. Would it be fair to say you've not served as a reviewer of any article that's had as its principal focus the maternal or fetal effects of --

A. In a peer review -- for a peer review journal?

Q. Yes.

A. Correct.

Q. All right. How about non-peer review journals; have you reviewed any?

A. There's a document that is not yet published called an "Asthma Continuum" for Texas Children's Hospital and the pediatric community at large that I'm a co-author on that addresses the issue of tobacco being a trigger for asthma.

Q. A symptom provoker?

A. Correct.

Q. All right. Is it your opinion that -- that that is the role, if any, of tobacco smoke in connection with children's asthma, that it acts as a symptom provoker?

A. It's a trigger.
Q. All right. Is there --
A. It's an irritant.
Q. Is there a difference between a symptom provoker and a trigger?
A. Well, I don't know what you mean by symptom provoker, and I do know what I mean by trigger.
Q. All right. A trigger means something that is --
A. Triggers the symptoms.
Q. Right. And not --
A. Probably the same.
Q. -- not as responsible for the underlying pathophysiologic change?
A. As an irritant and if it triggers, then by virtue of that association, it certainly causes symptoms, then it causes asthma.
Q. Asthma is a condition that can remain quiescent in a person until it's triggered by some environmental exposure, correct?
A. Correct.
Q. All right. And the underlying cause of asthma is something that you are differentiating from a symptom trigger,

correct?
A. Whatever that cause is.
Q. All right. And -- and are you here to testify that you have come to the conclusion that cigarette smoking causes asthma?
A. In that it causes the symptoms of asthma, it causes an asthmatic attack, yes.
Q. All right. Well, there are lots of things that can cause asthmatic attacks, correct?
A. Right.
Q. Down from cockroach allergens, to dog dander, to a laundry list of things that we could talk about for the rest of the day, correct?
A. And you may well.
Q. And -- and many -- many of those may have absolutely no role in causing the underlying clinical condition of asthma, correct?
A. They may not cause the cellular condition that results in the hypersensitivity, that those -- all of those agents, quote, "cause" an asthma attack or a worsening of airway and gas exchange. So depending on how you want to define it, they may not -- it may -- asthma may well be a genetically mediated disease. But once it's there, all of those

things, quote, "cause" an asthmatic condition.
Q. Have you ever smoked, Dr. Speer?
A. Yes.
Q. What did you smoke?
A. The brand?
Q. Cigarettes, pipes, cigars? What type of tobacco product?
A. Cigarettes.
Q. All right. For how many years?
A. Too many. Age 16 to about age 40.
Q. And how old a man are you now?
A. 55. I will be in October, at least. So I guess I'm close enough.
Q. And can you give us an idea of your smoking history? In other words, how --
A. What do you want to know about it?
Q. Well, were you an occasional smoker when you began at age 16?
A. Yes.
Q. All right. And by that, I meant an infrequent smoker.
A. Correct.
Q. All right. And when did you become -- when did you increase your -- what would have been

the frequency of smoking at age 16?
A. Oh, one, possibly two, possibly none in a given day, sometimes separated by no days, sometimes separated by three or four days.
Q. All right. And then when did that increase?
A. Let's see. I was a junior. Probably within the 12 months after I started.
Q. And what did it increase to?
A. I consciously kept my consumption less -- usually around a quarter of a pack, but sometimes up to a third of a pack a day. And in stressful situations, such as final exams, it went up to a pack a day. And then I would consciously try to cut it back.
Q. When you say you consciously kept it under a particular amount, was that because of health concerns?
A. Yes.
Q. What types of health risks particularly were you worried about?
A. As increasing evidence came -- well, you know, as increasing evidence came out in cigarettes' relationship to lung cancer and emphysema and having had relatives with both, I wanted to keep the numbers down to what I

considered to be reasonable numbers.
Q. And for how long a period of time -- did this phase of smoking went, as I understand it, from age 17 to -- to what age?
A. Age, about 26. Because after I got married, my wife said that "We're either married or you -- and you don't smoke in the house, or we're not married and you do smoke in the house." So that meant that smoking was confined to work and cups of coffee.
Q. All right. Did you find that the activity of drinking coffee and smoking cigarettes was often associated?
A. It was coupled.
Q. What kind of smoking history did you have after you got married at age 26 until the next time that smoking history changed?
A. About the same, except the consumption of cigarettes occurred at work as opposed to at home.
Q. Was it less?
A. No. About the same.
Q. All right. A quarter to a third of a pack?
A. Correct.
Q. All right. And that continued up until age

40?
A. Correct.
Q. All right. And what -- what caused you to quit?
A. I was beginning to have symptoms of shortness of breath, and I figured I had been stupid long enough. And so I cold-turkeyed one New Year's Day.
Q. All right. No smoking aids or anything?
A. No.
Q. No nicotine patches, no gum?
A. No.
Q. Just cold-turkey?
A. I wasn't happy, but no.
Q. All right. Have you smoked any since?
A. Well, you know, I fell off -- quote, "fell off the wagon" a couple of times over that three to -- about three-to four-month period of time. But I kept the same cigarettes. And there's nothing worse tasting than an old cigarette. It's just absolutely is appalling. Occasionally now I may have a cigar at a birth occasion or something like that, but rarely.
Q. What kind of cigars do you like?

A. Good ones.
Q. Me too.
A. But I don't buy them.
Q. Did your parents have rules against smoking when you were growing up?
A. No.
Q. You lost one or both of your parents to emphysema or lung cancer?
A. I lost my father to lung cancer --
Q. And --
A. -- and my grandfather to mesenteric infarction. And he was also having some pulmonary complications at the time of his death.
Q. Did you consider them at the time to be smoking-related diseases?
A. Lung cancer I did.
Q. All right. How about your grandfather?
A. In retrospect, yes. At that point in time, no, not necessarily.
Q. What -- what did your father do?
A. He was a career marine and then later an assistant golf pro.
Q. One of the two articles that you provide us as -- provided us as having reviewed, that

American Academy article, as I read it, it advocates the total elimination of cigarette smoking. Are you a person who believes that cigarette smoking should be totally eliminated by some sort of governmental intervention?

A. Not having been privy to the development of that statement, I would certainly say, as a personal opinion, that tobacco smoke is harmful to an individual. And I also believe that secondhand smoke can be harmful to given individuals. If people wish to smoke and are informed of all of the dangers of smoking, just like they can drink and be informed of the dangers of alcohol, then as long as it's only affecting them and not their children nor their fetuses, then -- or others, then fine. It's a little difficult to smoke and not do that. But if they wish to do it themselves, fine.

Q. Did you enjoy smoking when you smoked?

A. I don't know if it's enjoy. Got a high from the nicotine.

Q. And was --

A. It kept me awake.

Q. -- nicotine reinforcement, for lack of a better term, something that you enjoyed?
A. Yes and no.
Q. All right. What part of the yes part is there?
A. Until I got enough education to tell me that nicotine is addictive. And then I didn't like it.
Q. All right. And is that the no part?
A. That's the no part.
Q. All right. When did you -- when did you get enough education to realize that nicotine was addictive?
A. When I went to medical school.
Q. All right. And what was it in medical school that enabled you to make that determination? What did you learn?
A. I think it was a physiology course.
Q. Did you learn about the physiology of nicotine?
A. To a degree that was taught and that I subsequently remembered.
Q. Okay. And what was it about the physiology of nicotine that convinced you that it was addictive?

A. There are studies on addiction. That was the information. It's also, we learned, a potent vasoconstricting agent.
Q. The -- just so that we can -- make sure that I confine my questions to areas that are appropriate, are you an expert in pathology?
A. No, I'm not an expert in pathology. I know about pathology, but I don't hold myself out to be an expert in pathology.
Q. Okay. We've discussed statistics and biostatistics. How about molecular biology?
A. No.
Q. All right. And some of these may seem silly, but just bear with me. Because, I mean, certain witnesses have indicated knowledge or experience in particular areas that are not always predictable.
Medical ethics?
A. I think I'm fairly knowledgeable in the area of medical ethics, at least so far as it pertains to the newborn.
Q. And in the particular area of expertise, then, that you've referred to, is when it's appropriate to withhold or provide certain forms of medical treatments?

A. Correct.
Q. All right. Is there any area beyond that that you intended to refer to?
A. I don't believe so.
Q. Okay. How about psychiatry or psychology?
A. No.
Q. Pharmacology?
A. I'm knowledge -- I have knowledge regarding some issues of pharmacology but certainly not in very depth -- in great depth.
Q. Knowledge that you would expect a clinician to have who is --
A. Correct.
Q. -- prescribing pharmacologic drugs?
A. Correct.
Q. Okay. How about psychopharmacology?
A. No.
Q. All right. Toxicology? Again, in the area of a clinician who prescribes drugs.
A. Correct.
Q. All right. Consumer behavior in the areas of advertising or marketing?
A. Not an expert. I view with interest.
Q. We -- we all get bombarded with stuff on the TV. But other than that, would you -- would

you put yourself in the position of a layperson in terms of giving us opinions on advertising, marketing or consumer behavior?

A. From a perspective of observation as to what folks might do in the print and screen media and their effects on other folks, I think probably I have a bit more knowledge than the lay public, but I don't hold myself out as an expert in advertising techniques or quality thereof.

Q. Where -- where have you gotten your -- your additional or incremental knowledge regarding things pertaining to the print and screen media?

A. Well, it's -- it's the studies that have been reported widely regarding, for example, Joe Camel being recognized by first graders equal to or in greater numbers than Mickey Mouse. Obviously, Joe Camel is an advertising icon that advertises a product that small children probably shouldn't know about, necessarily, and certainly wouldn't -- you would not want them to use as they grow older.

Q. Have you made any study of whether or not there is smoking initiation or any smoking

behavior that has been reliably estimated with Joe Camel being one of the variables?
A. I think so. I don't have it at hand, but I'm pretty sure of having heard that there are such studies.
Q. Okay. Is that the extent of your awareness of any studies, if they exist, that you've heard that they exist?
A. I'm pretty sure they exist.
Q. Okay. But, I mean, beyond that, have you looked at the issue?
A. I have not looked at the studies.
Q. Do you know who the authors of the studies are?
A. No.
Q. Do you know where they were published?
A. They were -- the reading that I did about them was in the newspaper. And I think there were some editorials.
Q. Did you go to the studies themselves, or did you rely upon the newspaper account?
A. The newspaper and the editorials that were in the medical journals.
Q. You mentioned your own personal interest or experience in terms of the physiology of

nicotine. Do -- do you consider yourself an expert in substance dependence or addiction?

A. No.

Q. How about hospital administration?

A. I'm not a hospital administrator per se, although I am involved in various aspects of hospital administration so far as the development of policies, procedures, education, et cetera, of physicians, nurses, and other health care providers.

Q. All right.

A. So if you define that as administration, then I am an administrator. If you don't define it as administration, then I'm not.

Q. Okay.

A. You figure it out.

Q. Are you an expert in the history of the state of the art of any branch of medical science or any --

A. I'm sorry?

Q. Are you an expert in the history of the state of the art in any branch of medical science?

A. I'm not too sure I understand the question.

Q. Have you -- have you -- there are, believe it or not, medical historians whose professional

endeavor is to determine what the state of the art or the state of scientific knowledge was regarding particular issues at particular points in time. Are you an expert regarding the history of the state of the art of any branch?

A. Not as you just defined it. I'm a student of history, but not as you've defined it.

Q. All right. Are you an expert in medical economics?

A. No.

Q. Are you an expert in cigarette design manufacturing?

A. No.

Q. All right. Are you an expert in the area of environmental tobacco smoke exposure?

A. No.

MR. MINTON: Do you want to go ahead and change that? And we'll take a brief break.

THE VIDEOGRAPHER: The time is 3:06 p.m. We're going off the Record.

(A recess was taken)

THE VIDEOGRAPHER: The time is 3:15 p.m. We're on the Record.

Q. (By Mr. Minton) Dr. Speer, I think earlier this morning you told us you had not in connection with your opinions in this case made any sort of methodological review of the literature with respect to maternal smoking and adverse pregnancy outcome, correct?
A. Correct.
Q. In terms of writing down the opinions that were contained initially in Exhibit 1 and then in Exhibit 6, how did you decide what areas that you were going to -- to include in those documents?
A. I basically included those areas that I had knowledge within.
Q. All right. And that would have been the knowledge that you acquired by reading journal articles over the years?
A. And being at lectures and going to medical school, going through residency training, correct.
Q. All right. Would you be able to tell us with respect to low birth weight or small for gestational age, for instance, which were some of the well-designed epidemiologic studies?

A. As I said in response to your last question, the items that I wrote down on my report are those items that -- the knowledge of which I had acquired over a number of years. And I didn't go, as I also stated, to do a methodological research on which articles that knowledge came from in preparation for today.
Q. Are there, to your knowledge, specific studies which have looked at the relationship between maternal smoking and low birth weight with which you're familiar?
A. There have to be studies or else that's -- those statements wouldn't be in virtually every single textbook that deals with fetal or neo -- fetal medicine or neonatology.
Q. All right. And does that mean that -- that you don't know what those studies are?
A. As I have already stated, I did not do -- as you asked, I did not do any methodological research for this particular session. And those are statements that are contained in standard textbooks of neonatology and perinatal medicine.
Q. All right.

A. And if you would like, I can do such a research, but it might not serve your purpose.
Q. Do you know whether or not -- have you read any Surgeon General's reports with respect to smoking and health issues?
A. I've probably read the one -- at some point in time, the one that came out forcefully in support of the relationship of cigarettes being a causal factor in lung cancer, but that was a number of years ago, because I think it came out in 1990 --
Q. 1964?
A. -- or '64. Yeah. So, you know, I've read them, but I don't have them in my files. I did not review them specifically for today.
Q. All right. In terms of what, if any, statements the Surgeon General's report may have regarding maternal fetal issues, would it be fair to say you don't know?
A. Not as we sit here today, no.
Q. Okay. Have you reviewed any position papers of any of the societies that you belong to regarding what associations are believed to exist between maternal smoking and adverse

fetal outcome?
A. You noted the position paper that I provided you-all. I'm not too sure that this is a position paper, but I found it most interesting. This was sent to me today, and it appears to be on the American Academy of Pediatrics letterhead, so I'll offer it to you.
MR. BLEVINS: Just so the Record's clear, Doctor, that was not provided to you by -- by me or my law firm.
THE WITNESS: No. In fact, you were quite surprised that it existed.
Q. (By Mr. Minton) What is it?
A. What is what?
Q. This -- this document, as you understand it.
A. It appears to be a, quote, News Release and Press Statement from the American Academy of Pediatrics, Washington office.
Q. Okay. Who sent it to you?
A. The academy.
Q. All right. Through an E-mail or something?
A. I think they mailed that one.
Q. All right. Do you know the -- the occasion?
A. No. It was just, as it says on the little

piece of paper, "To Dr. Speer from Sue Tilez, FYI check, Advise comment, no check, please return, no check."

Q. All right. Who is Sue?

A. She's the administrative staff person assigned to the American Academy of Pediatrics Committee on Fetus and Newborn.

Q. Okay. And she knew you were testifying in this case?

A. No.

Q. This was --

A. Unsolicited.

Q. Okay. Is there something of significance in this document?

A. Well, I think so. I was struck by the paragraph -- not the bottom paragraph, but the next. It states: "Incredibly, as estimated, the elimination of smoking would reduce infant deaths by 10 percent and decrease the incident of low birth weight by as much as 25 percent," which I thought was germane to our discussion today.

Q. Do you know the nature of that calculation?

A. No.

Q. All right. Do you know what a population

attributable risk is?
A. I'm sorry?
Q. Do you know what a population attributable risk is?
A. No. I'm not familiar with that definition.
Q. All right. Do you know what data may or may not have been consulted in terms of the formulation of that statement in that document?
A. No.
Q. Its accuracy is something that you've investigated?
A. Well, the academy is not -- not in the habit of publicizing on a national basis inaccurate statements, so I would anticipate that the accuracy is quite tight.
Q. Has the American Academy of Pediatrics come out in favor of the elimination of cigarette smoking?
A. You've already stated that they did.
Q. Is that consistent with your understanding?
A. Correct.
Q. Is the American Academy of Pediatrics a governmental institution?
A. No.

Q. All right. Is it a -- an institution that has a certain amount of politics attached to it?
A. Its only politics appear to be the welfare of children.
Q. As perceived by the people who happen to be the leaders of the American Academy of Pediatrics?
A. No. By the public at large.
Q. Do you know who drafts news releases and press statements for the American Academy of Pediatrics?
A. This one appears to be authored by Dr. Murial Wolf, the president of the D.C. chapter of the American Academy of Pediatrics.
Q. Okay. Do you know anything about Murial Wolf?
A. No.
Q. Dr. Speer, what I'd like to do is go back. You had mentioned for us some specific health endpoints. And I'd like to begin somewhat out of order in the ones that we discussed earlier.
But with respect to premature births, do you or your -- the people that you work with

at any of the five hospitals that you're on staff here use any sort of clinical risk prediction scale for a premature delivery?

A. Could you define what you mean by a prediction score? Are you talking about do we have methodology that implies to us at a given gestational age and weight what the outcome -- survival outcome will be?

Q. No. There's a -- there are a number of clinical risk prediction scales that some practitioners use in terms of -- of associating risk factors with the -- the clinical outcome of interest, and in particular in this case, premature delivery. Bob Creasy has developed one.

A. Right, but those are all obstetrical risk scores. Remember, I get the baby after it's born, not before.

Q. All right. Are -- is -- to your knowledge, are any of those clinical risk prediction scores being used by obstetricians in the hospitals in which you practice?

A. I have no independent knowledge that they are or they are not. We have perinatologists that function in many of these -- many of the

hospitals that I noted, and they may well use risk scores. But I can't tell you if they do or they don't.

Q. All right. Do you know what the predictive capability is of any of the clinical risk prediction scales that are used by OB-GYNs for the health endpoint of premature delivery?

A. Why should I? I'm a neonatologist.

Q. And -- and the reason you wouldn't be interested in that is because --

A. I have no influence on the care of the pregnant mother.

Q. All right. Or the -- the factors which seem to or do not seem to predispose to -- to particular maternal fetal health endpoints?

A. I'm not too sure I understand your question.

Q. Well, let's go back to the one I asked.

A. You've asked several.

Q. Do you know if any clinical prediction -- risk prediction scales are used for prematurity by the OB-GNs -- OB-GYNs with whom you practice at the five hospitals where you're on staff?

A. I've answered that.

Q. You don't know?
A. Right.
Q. Okay. Do you know what the predictive capability of any clinical risk prediction scale for prematurity is?
A. No.
Q. All right. Can you give us a list of clinical conditions that you -- that, in your opinion, are associated with premature delivery?
A. I can give you an incomplete list. I would refer you to an obstetrician for a more complete one. Young age, old age, so far as maternal age. Younger than -- in other words, younger than 19 and older than, say, 35. A history of smoking. Incompetent cervix. A history of prior pre-term delivery. Occult or non-occult bacterial infection, particularly urinary tract infections, but also infections involving the amniotic membranes, fetal membranes. Car accidents. The trauma -- blunt trauma to the uterus or other forms of blunt trauma to the uterus. Abruptio placenta caused by either idiopathic etiologies or cocaine use, or

tobacco has been associated. Chorioamnionitis I covered in the occult or non-occult infections. There are undoubtedly some that I'm leaving off, but those are probably the major ones.

Q. All right. You mentioned with respect to abruptio placentae that there had been some literature which had associated placental abruptions with tobacco usage. Have you investigated the nature of that association in terms of evaluating the likelihood that it's causal?

A. In each patient, so far as investigating whether their particular abruptio is caused by tobacco? Is that your question?

Q. That wouldn't be possible, would it, Doctor?

A. It would be rather difficult in an individual case.

Q. No. My question was, doing some sort of investigation of the literature to determine whether or not -- you used the word "association." And I guess what I was getting to is whether that word was used in the sense that there has been noted a statistical relationship between maternal

smoking and the occurrence of placental abruptions or whether or not you had investigated the nature of that statistical association and were prepared to come forward and say that, in your opinion, that was a causal association.

A. How are you defining "causal"?

Q. Well, that's a good question. Let's go back and back up a few steps.

Are all of the -- are all of the conditions that you've covered in Exhibit 6 that we have mentioned multifactorial?

A. There are multiple causes for all of those conditions, correct.

Q. All right. For any of those multiple causes, have you made an analysis of which are -- which occur more frequently on a population basis?

A. I personally have not.

Q. Have you, with respect to any of those multiple causes, looked at the strength of the association that has been said to exist?

A. As far as doing research on my own or perusal of the literature?

Q. Yes.

A. No.
Q. For any of the multiple causes of the conditions that are identified in your disclosure statement, Exhibit 6, you have not investigated what the relative risk of that factor was in any population?
A. Correct.
Q. And would it therefore follow that you have not sought to rank top to bottom the strength or relative risk of any association that is a quote/unquote "multiple cause" of any of those conditions?
A. Multiple cause or a single cause? If you're going to rank, you have to almost have single causes.
Q. All right. Are any of them -- have any of them been identified to be single causes?
A. If you have multifactorial causes each of which that is independently associated with the result, then it is a cause.
Q. In the sense that it was present?
A. More often than not, than absent.
Q. All right. Let me -- let me give you this hypothetical, Doctor. There is a an association between black race and low birth

weight outcome, correct?
A. Correct.
Q. There's an association -- a statistical association between cigarette smoking and low birth weight outcome, correct?
A. Correct.
Q. There's a statistical association between low socioeconomic status and low birth weight outcome, correct?
A. Depending on the population.
Q. Well, you wouldn't find it in a high socioeconomic status population, but when you --
A. Well, actually, in a high socioeconomic black population, you have a lower birth weight than you have in a white high economic -- socioeconomic population, so it's an independent.
Q. All right. So it is an -- it's an independent risk factor?
A. Race, yes.
Q. And -- well, and low socioeconomic status is as well, is it not?
A. It depends on the population group. You have very few -- you don't have small babies for

gestational age, you know, in the Latin-American population, you know, not nearly as many.

Q. Okay. But --

A. They tend to be big babies. And they certainly are low socioeconomic.

Q. The Latin?

A. Uh-huh.

Q. The ethnic groups?

A. Uh-huh.

Q. All right. So it is -- it can be present or absent, depending upon the particular population that's being investigated? Socioeconomic status seems to mediate an effect --

A. Hang on. Hang on. Are we still talking about low birth weight?

Q. Yes.

A. Okay.

Q. Socioeconomic status seems to mediate an effect in certain ethnic populations but not in others?

A. If you're stating that there are more pre-term infants born and that's where you're coming for low birth weight, it's probably a

true statement.
Q. All right. And there is a component of low birth weight which simply cannot be explained by the presence of any risk factor, correct?
A. At present.
Q. All right. There are -- there are women who, although they appear to have no predisposing factors for a low birth weight baby, nonetheless have them, correct?
A. They are a segment of the population, correct.
Q. All right. And so if a woman came into your office who was black, of low socioeconomic status, a smoker, there would be no scientific means of determining which, if any, of those risk factors alone or in conjunction with one another caused that low birth weight outcome, correct?
A. Except that one of those you could intervene in.
Q. I'd like you to try and handle the hypothetical that I gave you, Doctor.
A. The hypothetical may well be that cigarette smoking is the cause of your low birth weight patient. So if the mother stopped smoking

and the baby's fetal growth improved, then that would be circumstantial evidence that indeed in that particular hypothetical that the cigarette smoking was the cause of the observed fetal low birth weight. If, on the other hand, the mother smoked -- and this is assuming we achieved that early enough in the pregnancy and we're not toward the end of pregnancy. But in your hypothetical, if we indeed found that that was the case, then I think you could make a strong case for cigarettes. On the other hand, if fetal growth did not improve after smoke -- after cessation of tobacco smoking, then you certainly would be, I think, reasonable in figuring that there might be another etiology in that particular hypothetical.

Q. I think you misunderstood the hypothetical that I gave you, and that is a woman who has already given birth to a low birth weight baby.

A. You didn't say anything about birth, sir.

Q. Oh, I'm sorry. The birth has occurred.

A. Okay.

Q. All right. And she has given birth to a low

birth weight baby.
A. Okay.
Q. And she is black, of low socioeconomic status, and a cigarette smoker.
A. Okay.
Q. There is no scientific means by which to decide which, if any, of those risk factors caused that outcome in that particular individual, is there?
A. At present, I would agree with you.
Q. Nor is there any way -- even if we were to assume that all three combined in some way, there is no way at present to -- to give the relative contribution of each of those risk factors in that particular individual, correct?
A. Quite so. It's a single patient.
Q. All right. And the same is true for every single health effect about which you have referred in Exhibit 6, correct? Where there were multiple risk factors present, we do not have the present ability in an individual to determine which, if any, of those risk factors caused that outcome or if they all caused it, what part they played, correct?

A. Once the baby is born.
Q. Right.
A. I think that's probably true.
Q. There's nothing pathognomonic that will tell us, is there?
A. Unfortunately, no.
Q. All right.
A. Not once they are born.
Q. As I understand your report, in terms of the low birth weight outcome -- and we'll confine ourselves to that, small for gestational age and low birth weight. And I -- the look you just gave me indicates that I've already injected something confusing into the question.
A. Yeah.
Q. Did I overread that?
A. Uh-huh.
Q. Okay. We're confining ourselves to small for gestational age and low birth weight.
A. Why don't we confine ourselves to either one or the other? Because, as I've already pointed out, SGA is low birth weight.
Q. Okay.
A. Now, if you want to talk about a subset of

low birth weight infants who are SGA, I'm perfectly comfortable there; or if you want to talk about all low birth weight infants, I am fine there.

Q. Okay.

A. But it's difficult for me to lump the two.

Q. All right. What I'm trying to do is distinguish it from prematurity.

A. Prematurity in many instances equates with low birth weight.

Q. Right. Well, we'll stick with term "low birth weight" at this point. Okay?

A. Okay.

Q. All right.

MR. MINTON: Off the Record.

(Discussion off the Record)

MR. MINTON: Back on the Record.

Q. (By Mr. Minton) Doctor, quite frankly, I forgot what I was going to ask you, so I'm going to go on to a different topic.

In terms of -- was the answer you gave about no pathognomonic sign or symptom, does that cover the gamut of maternal fetal effects that are mentioned in Exhibit 6?

A. Well, if you're talking about path -- using

the word "pathognomonic" to be a single clarion moment where you say eureka, probably.

Q. Okay.

A. If you're talking about something, for example, like thrombocyte -- not thrombocytopenia, but polycythemia as being an -- a strong indicator of intrauterine hypoxia, then it almost falls into the eureka category. You can't necessarily say what caused the hypoxemic state, but you can say there was a hypoxemic state.

Q. All right.

A. So I'm not too sure whether that clarifies or confuses.

Q. Well, I need to narrow it some. With respect to maternal smoking as being a risk factor or one of the potential causes that were present in a particular individual, there's not -- there's no sign or symptom post-birth which of itself points back to smoking as a cause, does it -- is there?

A. With the exception that if you looked at the placenta, looked at the maternal history and you only had smoking, there may be a few

instances where you could point the finger at intrauterine hypoxemia and say that smoking is more likely than not the cause.

Q. All right. That would be the circumstance that you can think of?

A. That would be a strong association.

Q. All right.

A. You can also take a look at placentas, again, if smoking is sort of the single risk factor that is present in a given pregnancy and point the finger towards fetal effects.

Q. Okay. I -- but I -- what I tried to do was confine my question to an instance in which there were multiple risk factors present. And with that qualification, would it be correct to say there are --

A. When there are multiple risk factors present, it is difficult to single out a unique risk of one risk factor versus another.

Q. Okay. Well, we can't do it, can we?

A. Not yet.

Q. Okay. You mentioned placental and fetal hypoxemia. Is there a characteristic change in the villus structure which is found in babies who have chronic hypoxemia?

A. Are you talking about babies who have multiple -- mothers who have multiple risk factors or a single risk factor?
Q. Multiple risk factors.
A. No.
Q. All right. And are you suggesting that there is a pathophysiologic change which occurs in the placental structure which has been uniquely associated with cigarette smoking?
A. Not uniquely associated.
Q. As a general statement, Dr. Speer, would you agree that, as confirmed through placental pathology, very low birth weight pathology is principally where the villus structure does not form properly?
A. Are we talking about term, preterm?
Q. Both.
A. And you're talking in what circumstance?
Q. In terms of pathophysiologic change that can be noted in very low birth weight babies, is it correct to say that the -- the central pathophysiologic change that is noted is the failure of the villus structure to form correctly?
A. For all very low birth weight babies?

Q. Not -- that may be overstating it. But the principal or the cardinal or the most common pathophysiologic abnormality that is found.
A. In just everybody?
Q. No. In very low birth weight babies.
A. Right. But I mean everybody within the rubric of very low birth weigh babies?
Q. Yes.
A. You're asking is villus abnormalities a common finding in that population?
Q. Yes.
A. Yes.
Q. And does that -- does that -- is the mechanism by which that occurs, in other words, the villus structure fails to form properly, is that because the -- the second wave of trophoblast does not come down and alter that villus structure to change it to accommodate the higher levels of uteroplacental blood flow?
A. Remember, I'm not a pathologist. You established that. And so I would leave that answer to a pathologist.
Q. All right. Do you know whether that abnormality is the central factor in those

pregnancies in which there is a reduced maternal blood flow to the placenta?

A. Well, so far, we haven't really established what kind villus abnormality we're talking about, because there are multiple villus abnormalities. And, again, so far as the specific incidence and risk of villus abnormalities, I would bow to the expertise of a placentologist.

Q. Well, let's walk through step by step, then. Is it your understanding that in order for the spiral arteries of the placental bed to change into proper uteroplacental vessels that there have to be two invasions of cytotrophoblastic cells?

A. I've already stated that I am not a pathologist, and I'm not an -- I don't deal with issues of anatomy.

Q. Right.

A. I am a neonatologist. It is my understanding that there are placental changes, included of which villus changes, in patients who deliver premature babies. And there's an association of some of those villus changes with smoking mothers.

Q. Okay. But whether or not the villus changes that are associated with very low birth weight outcomes are the type of villus changes that have been associated with the placentas of smoking mothers, you're not here to say, I take it?
A. I believe they are in some instances. But I'm not here to say exactly what those changes are.
Q. Okay. In an abnormal placenta, what are considered evidence of a growth restriction? What findings are considered evidence of a growth restriction?
A. A growth restriction of what?
Q. A growth restriction of either the placenta or the fetus.
A. Well, that's why I asked.
Q. Okay.
A. Now, let's go back and ask the question again. And tell me which one you want me to respond to.
Q. In an abnormal placenta, what is considered evidence of a growth restriction?
A. Placental weight is the common one. Placental changes, including fibrosis,

infarcts, increased calcification for age and specific changes that -- I'm not a pathology and can't tell you exactly what they are.

Q. Are infarcts or calcification signs of a persistent continuous vasoconstriction?

A. They have been associated with that.

Q. In other words, they have been found to be present where persistent continuous vasoconstriction has also been found to be present?

A. Correct.

Q. Have they been found to be present where continuous persistent vasoconstriction has not been found to be present?

A. Probably.

Q. Is acute athyrosis an abnormality of the maternal uteroplacental vasculature that's considered to be of central importance in placental pathology?

A. Athyrosis --

Q. Yes.

A. -- or sclerosis?

Q. Atherosclerosis.

A. That's not -- I have not -- I have not heard that term used in the description of

placental pathology by our pathologists, so I can't answer whether it could be or not. They don't use that term, so I can't tell you.

Q. How does chorioamnionitis mediate a premature delivery?

A. Presumably by the elaboration of chemical by-products that stimulate uterine muscle contraction.

Q. Do we -- do we know what the mechanism of premature labor is?

A. So far as the prostaglandins and kinins and all of that business that do what results in premature labor?

Q. What the hormonal process is that --

A. Not totally, no.

Q. Is -- is cigarette smoking negatively associated with maternal hypertension?

A. Negatively associated? You mean it causes or it doesn't cause? How are you using "negative," I guess is the question?

Q. Is it -- well, is maternal hypertension found to be less prevalent in mothers who smoke?

A. I don't know. I know that in the general population of people who smoke, hypertension

is more often associated with people who smoke than people who don't smoke.

Q. All right. There's a difference between pregnancy induced or maternal hypertension and generalized hypertension, is there not?

A. There is a difference between pregnancy-induced hypertension or preeclampsia or other -- a chronic form of hypertension. And my knowledge base associates the chronic form of hypertension to smoking, not necessarily the pregnancy-induced hypertension.

Q. All right. So you're not here to tell us that cigarette smoking is associated with maternal hypertension?

A. Correct.

Q. All right.

A. No, I'm not here to tell you that it's associated with a pregnancy-induced maternal hypertension.

Q. All right. It may or may not be associated with nonpregnancy-induced maternal hypertension?

A. I think there is a good association of that.

Q. All right. The -- is there a negative

association between maternal smoking and eclampsia and preeclampsia? In other words, are those conditions found to be less prevalent in mothers who smoke?

A. I don't know. I would refer you to an obstetrician for that.

Q. Now, you have in your disclosure statement a number of statements regarding, for lack of a better term, heme synthesis or effects on heme precursors, and specifically carbon monoxide binding to hemoglobin to form carboxyhemoglobin, correct?

A. Uh-huh, yes.

Q. Is -- would it be correct to say that the critical issue, if one were to hypothesize, maternal smoking as a mechanism for a fetal effect mediated through carbon monoxide ingestion would -- and that effect being lodged in the fetus, would it be correct to say that the issue of critical importance would be an actual disruption in fetal blood gases?

A. I'm not too sure of your question.

Q. Let me -- let me try and break it down some.

A. Because fetal blood gases are not necessarily

going to tell you what you want to know. What you want to know is oxygen carrying capacity. And carbon monoxide does interfere with oxygen carrying capacity.

Q. There are differences between maternal oxygen carrying capacity and fetal oxygen carrying capacity, correct?

A. As a general statement in the de novo condition?

Q. I'm sorry?

A. As a general statement in the de novo condition?

Q. Yes?

A. Correct. You've got different types of hemoglobin.

Q. Different types of hemoglobin. You have different levels of erythrocyte production. You have a different stroke rate from the heart. You have varying degrees -- a varying number of issues which impact actual oxygen carrying capacity in blood gas transfer, correct?

A. As a general global statement, correct.

Q. And the hypothesis that has been attached to carbon monoxide in terms of its, you know,

potential for interrupting -- I mean, what we're interested in in terms of investigating carbon monoxide as a component of cigarette smoke when a woman smokes is whether or not it changes the level of oxygen that reaches target tissues in a fetus, correct?

A. Correct.

Q. All right. And --

A. Which is oxygen carrying capacity.

Q. Well, which is actual blood gas levels, correct?

A. No. It's oxygen carrying capacity. Because oxygen carrying capacity is quite rightly, as you pointed out, based on the number of red cells, the oxygen tensions and the interference or noninterference with other gases insofar as oxygen attachment to the hemoglobin moiety.

Q. Well, is -- if -- if the critical issue, as I think we've agreed, if it is actual delivery of oxygen to the target cells in the fetus --

A. Correct.

Q. -- is that not going to be a function of fetal blood gas levels?

A. Not necessarily, because you may not measure

with fetal blood gas levels the amount of oxygen that's being carried in a baby who has carbon monoxide and carboxyhemoglobin.

Q. If that's one of the blood gases you look for, wouldn't that tell you?

A. Not necessarily. It depends on what your red cell mass is.

Q. So --

A. Oxygen carrying capacity is higher in a patient who has a hemoglobin of 15 as opposed to a patient who has a hemoglobin of 10, and the blood gasses can be exactly the same.

Q. Right. But the partial pressure of oxygen is the critical issue, is it not?

A. No. It's the oxygen carrying capacity of the hemoglobin, and the partial pressure of oxygen only has a partial bearing on that. The partial pressure of oxygen in a measured sample may be exactly the same in a patient with a hemoglobin of 15 versus a hemoglobin of 2. But the oxygen carrying capacity is changed by a factor of whatever that is, 20 or so -- more than that.

Q. Okay. And the carboxyhemoglobin issue concerns --

A. Sorry.
Q. That's okay. Carbon monoxide binds preferentially to heme compared to oxygen, correct?
A. Correct.
Q. And heme is the means by which oxygen is transported in the bloodstream, correct?
A. Correct.
Q. And the extent to which carbon monoxide is bound to heme, forming carboxyhemoglobin, affects the oxygen carrying capacity of the blood, correct?
A. Correct.
Q. And carboxyhemoglobin is not as able to carry -- to transport oxygen as oxyhemoglobin, and so therefore the partial pressure of oxygen would fall, correct?
A. No. You've got two different -- you've got two different scientific principles. You've got the dissolving of oxygen within the fluid, which is the partial pressure. And then you have the oxygen carrying capacity of hemoglobin, which is a different concept than the partial pressure of oxygen. And we go around and around with this with our

residents and fellows because there is a distinct difference.

Q. Okay. Are the light absorption characteristics of fetal carboxyhemoglobin the same as adult carboxyhemoglobin?

A. I don't know.

Q. Do you know whether or not if you looked at fetal blood using light absorption characteristics that pertain to adult carboxyhemoglobins whether or not you'd get spuriously high results?

A. I don't know. I know that the usual oxygen saturation probes that have the wave length that's adapted for adult hemoglobin give erroneous results or at least they are not as accurate as those that have been adapted for fetal hemoglobin.

Q. So if you used a blood gas analyzer on fetal blood and looked at the level of carboxyhemoglobin, you'd be likely to get inaccurate readings if you used adult hemoglobin as your standard of measurement?

A. That's my understanding. But, again, I'm not a pathologist or a constructor of those types of equipment. And usually I don't have a

fetus, so I can't really tell you.
Q. Do you --
A. We don't give carbon monoxide to our babies once they're born.
Q. No. No. But do you do chordocentesis on --
A. No, because I'm a neonatologist, not an obstetrician or perinatologist. I know that confuses some folks, but there is a difference. I'm just a GP of babies.
Q. Have you -- have you looked at any studies to determine -- first of all, let me ask you, is the statement about the possibility of -- strike that.

Should we interpret the statement in Exhibit 6 that you've made about carbon monoxide as Dr. Speer's opinion on the mechanism by which maternal smoking mediates adverse effects in the fetus, or is it suggested as a hypothesis?
A. The statement that carbon monoxide binds preferentially as compared to oxygen to hemoglobin is a true statement. The fact that the fetus already is in a relatively low oxygen environment is also a true statement. If the amount of oxygen in a given particular

fetus is compromised by any condition, the fact that you have carbon monoxide hemoglobin that's taken out of the ability to carry oxygen to tissues is a true statement.

The inference would be that a reduced oxygen environment is not a good one, which would be also a true statement.

Q. All right. And all of those are stated as -- as assumptions building on one another. Is it your opinion, however, that the mechanism of a particular adverse pregnancy outcome is mediated by increased carboxyhemoglobin, which causes relative hypoxia?

A. Am I stating that that is a cause for all pregnancy injury to the fetuses?

Q. For any in particular.

A. No, I'm not saying it's a universal insult. I am saying it is an insult.

Q. All right.

A. It is a cause.

Q. And are you saying it is a cause of a particular outcome?

A. I would think that it explains some of the outcomes that have been observed because it is known that carbon monoxide is increased in

smokers; it is increased in fetuses. And carbon monoxide causes those things that we've just talked about and does decrease the amount of oxygen that can be carried by red cells. I am certainly not saying it is an only cause.

Q. All right. Are high hemoglobin levels in neonates a characteristic consequence of chronic fetal hypoxemia, then?

A. Yes. Apparently, it is.

Q. And if chronic hypoxia were the cause of smoking-generated effects, whether it be fetal growth, retardation or some other effect, would not one expect birth weights to decrease with rising hemoglobin levels in those neonates?

A. Birth weights increase?

Q. We'll go back. We'll go back.

A. Please. I'm a simple person.

Q. Okay. If -- I think we agreed or you agreed with the statement that high hemoglobin levels in neonates would be a characteristic consequence of chronic fetal hypoxemia.

A. That has been found, yes.

Q. All right. And let's -- let's confine the

discussion now just to low birth weight rather than any other endpoint.

A. Okay.

Q. If chronic hypoxia were the cause of smoking-generated fetal growth retardation, wouldn't you expect birth weights to decrease with rising hemoglobin levels in those neonates?

A. Not necessarily.

Q. Why not?

A. Because if you -- if you're smart enough to increase your hemoglobin level to obviate the effects of the carbon monoxide, your growth may proceed a pace, at least based on the lack of oxygen. But there's -- growth is affected by other nutrients, such as glucose, fats, proteins, that cross the placenta.

Q. Well, if carbon monoxide was the -- the mechanism by which birth weight reduction was caused and carbon monoxide -- increased carbon monoxide consumption by the mother --

A. Consumption?

Q. Well, exposure.

A. Okay.

Q. If the mother was a smoker and was exposed to

carbon monoxide -- and we've established that carbon monoxide would result in high hemoglobin levels in the neonate, correct?

A. Keep going.

Q. Then in neonates with reduced birth weights, wouldn't we expect to see higher levels of hemoglobin?

A. Infants with reduced birth weights, which we haven't established where the reduced birth weights come from, you're proposing that their birth weights would be further reduced if they had high levels of red blood cells, correct? That's -- am I quoting you correctly?

Q. No. I -- let me start over. If -- if we found a positive association between maternal smoking and high hemoglobin levels in the fetus, which we've found, correct?

A. Yes.

Q. And that's -- that is in the scientific literature, correct? That's a fetal response to maternal exposure to carbon monoxide, correct?

A. That is hypoth -- that is the hypoth -- that is the sequence that is thought to occur,

yes.
Q. All right. And -- and -- but it's a demonstrated fact that, for whatever reason, the fetuses of mothers who smoke have higher levels of hemoglobin, correct?
A. Okay. Okay.
Q. All right. If smoking causes higher levels of hemoglobin -- maternal smoking causes higher levels of hemoglobins in the fetuses of mothers who smoke, and if smoking -- and that is mediated through carbon monoxide exposure, and if carbon monoxide exposure was felt to be the mechanism for reduced birth weight --
A. Which I'm not too sure it is.
Q. Okay. That was the only point that I was endeavoring to establish here.
A. Why didn't you just ask me?
Q. All right. You are not here to say that carbon monoxide exposure is a mechanism by which low birth weight is produced?
A. No.
Q. Okay.
A. But it can cause injury in its own right.
Q. All right. And are you here to say that

carbon monoxide exposure through maternal smoking causes any of the effects that are listed on Exhibit 6?

A. I don't know. We know that instances where you have evidence of chronic intrauterine oxygen deprivation, you have a higher associated risk of poor intellectual outcome. So whether or not that will ultimately be proven in this instance or not, I'm not prepared to say. But certainly we know that there are a variety of models where chronic hypoxia is induced to either in the animal model or observed in the human model where those animals and/or humans aren't quite as bright as they should be.

Q. Okay. As I understand it, however, in -- at least in the context of low birth weight, you are not providing an opinion that says maternal smoking causes a change in fetal carboxyhemoglobin which results in hypoxia, which results in low birth weight?

A. Correct.

Q. All right. Are you proposing that nicotine acting as a vasoconstrictor is a mechanism by which low birth weight is produced?

A. It may well be.
Q. All right. It is a possibility?
A. It is an action of vasoconstricting agents to cause problems with placental blood flow and hence nutrition of the fetus --
Q. All right.
A. -- whether it be nicotine, cocaine or others.
Q. Nicotine, as a vasoconstricting agent, has been hypothesized as a possible mechanism by which maternal smoking might mediate certain adverse pregnancy outcomes. Are you saying that it does?
A. I think it does, yes.
Q. Which outcomes?
A. I would say probably related to the small size. It may be related to the rupture of the placenta issue. It may be related to the premature labor issue. And it may be related to the adverse intellectual outcome that's recently been reported if that study is substantiated.
Q. All right. One of those you said, "I think it does." The remaining you said, "I think it may." Are we to infer from that that the ones you said "I think it may," that those

you consider to be unproven, but the one you said "I think it does," you consider to be proven?

A. Okay. Maybe I misspoke. So far as prematurity, abruptio placenta, small size, I think it does. So far as the intellectual disability that was reported by our friends that you know the name of and I keep forgetting --

MR. BLEVINS: Drewes.

THE WITNESS: Huh?

MR. BLEVINS: Drewes.

THE WITNESS: Drewes.

A. -- it may, because that's a single study.

Q. (By Mr. Minton) Do you know the means by which nicotine acts of as vasoconstrictor?

A. At one point in time, I could quote you the physiology and the end organ receptors, but at this point in time, no.

Q. All right. Have you seen -- have you seen data in either animals or human beings which has sought to measure either the -- the diameter change in any vessel or the flow rate change in any vessel which has occurred as a result of -- I've got to start over.

Have you seen any animal or human data which sets forth the caliber or diameter change in any vessel connecting the mother to the fetus or a flow rate change in any vessel connecting the mother to the fetus in which the authors have sought to characterize the change in flow occasioned by nicotine ingestion or absorption?

A. I think I remember reading such, but it's been a number of years ago. And once again, I would refer you back to the obstetrical folks because that's their area of interest and expertise.

Q. All right. Is there overcapacity in the uteroplacental vessels in terms of their normal physiologic function?

A. What do you mean by overcapacity?

Q. Is there -- well, in terms of vessel diameter, is there more vessel diameter there than is necessary in order to effectuate blood flows to maintain fetal blood gas levels above a state of hypoxia?

A. In the normal pregnant woman during most of pregnancy, particularly in the early part of pregnancy, it's my understanding that blood

flow available to the developing fetus is more than the fetus needs. However, toward the end of pregnancy, that is not necessarily the case. And, indeed, that is one reason why obstetricians do not wish their mothers to go beyond 42 weeks, because the fetal requirements outstrip the ability of the placenta to maintain those requirements.

So at certain points of pregnancy, your statement is undoubtedly true. However, at other points in pregnancy, it does not appear to be true.

Q. All right. At what points would vasoconstriction occur in a fetus?

A. At what point in time would a chemical cause vasoconstriction in a fetus?

Q. No. At what physical locations would we be concerned about vasoconstriction?

A. I'm unsure as to what you mean by physical locations.

Q. All right. There -- there is a uteroplacental vessel, which is the major means of --

A. There's the uterine artery. Okay. There's the uterine vein. There's the uterus.

Q. All right. And are we concerned about a site of vasoconstriction there?
A. We certainly could be.
Q. All right. Are we concerned about sites of vasoconstriction elsewhere?
A. Depending on what you're looking for. If you're looking at the delivery of nutrients to the fetus, vasoconstriction on either side, the maternal side or the fetal side of the placenta would be deleterious to the fetus. If you're talking about vasoconstriction to mesenteric arteries or to the brain, then it's vaso -- fetal brain, then you're talking about vasoconstriction in those areas. So I'm not too sure what you're driving at.
Q. Well, blood is flowing through a variety of different vessels between the mother and the fetus, correct?
A. No.
Q. How many vessels does it flow through?
A. Blood does not flow from the placenta to the fetus. Blood is within the sinusoids of the placenta. It's picked up by the fetal vessels, if you will, of the placenta and

then carried via the umbilical vein back to the fetus. There's not a connection between the maternal circulation and the fetal circulation. It's diffusion of oxygen across.

Q. The fetal membrane?

A. The membranes, right, that comprise the placenta, which is why normal oxygen tensions in the fetus are lower than the oxygen tensions found in the mother.

Q. All right. And which -- which raises an issue with respect to vasoconstriction, does it not? I mean, where -- where are you positing vasoconstriction --

A. Well, if vasoconstriction occurs in the uterine artery, then you're not going to get as much blood to the maternal side of the placenta.

Q. Okay.

A. If you're hypothesizing vasoconstriction on the fetal side, you're not getting fetal blood to that area of the placenta to pick up oxygen. So you can have vasoconstriction on either side of the placenta that may be unwise.

Q. Do you know how potent a vasoconstrictor nicotine is?
A. It's supposed to be pretty vasoconstrictive.
Q. All right.
A. It's supposed to be one of the most potent.
Q. Rated against what other vasoconstrictive drugs?
A. I can't remember the table that we were shown way back, way back, but it's fairly high up on the list. I can't tell you exactly what all that they looked at at the time that I was being taught that.
Q. All right. Are there -- are --
A. But I know it's one of the benchmarks for vasoconstriction in many studies.
Q. All right. Are there physiologic occurrences in human beings who smoke that are in response to vasoconstrictive effects to maintain circulation?
A. I think you just lost me on the last phrase.
Q. Okay. Are there changes which occur subsequent to vasoconstriction in order to maintain an oxygen balance in a person who smokes?
A. Again, I'm not too sure where you're coming

from. You can get -- you get vasoconstrictive changes in people who smoke.

Q. Are there -- are there, then, other changes which occur which moderate the impact of that vasoconstrictive effect?

A. If you're talking about increased contractility of the heart, yeah.

Q. Anything else?

A. I'm not too sure where you're headed. I mean, you have constriction of -- you can have arterial constriction in people who smoke, which is thought to be one of the reasons that long-term smoking is associated with hypertension. The physiologic response to that is increased stroke volume and contractility of the heart, which, in turn, can also contribute to hypertension. You can get vasoconstriction of abdominal arterial vessels, and you can get vasoconstriction of coronary vessels, which, depending on the blood supply and atherosclerosis and other factors, may be deleterious to patients. I'm not too sure where you want me to go with this.

Q. Does that --

A. You've got a global question hanging out there, and I'm looking at the Milky Ways.
Q. Does that -- does that vasoconstriction occur uniformly throughout all veins and arteries in the body, or is there some partitioning which occurs?
A. There is probably partitioning. But once again, you're getting away from my field, which is babies, and getting into adults, which remember for a neonatologist, anybody over 28 days of age is an adult. In fact, it's a geriatric patient as far as I'm concerned.
Q. Have you seen any animal studies on what toxicologists have done when they've administered nicotine to animals to determine effects in the placenta or in the uterus?
A. Perhaps a long time ago, but I have not recently.
Q. All right. Would you think that that would be a source of data that one ought to consult in order to evaluate the likelihood that nicotine, through its action as a vasoconstrictor, may or may not mediate adverse pregnancy outcomes of the fetus?

A. It's certainly reasonable to use an animal model to assess the effects of nicotine.
Q. Would -- would the use of an animal model be reasonable to assess the effects of any constituent of tobacco smoke?
A. It would be a useful first step. Unfortunately, animals are not humans. So animal modelling doesn't always predict what will happen in the human model.
Q. All right. Are there -- well, let me -- let me ask you in terms of your own causal evaluations. How important to you are animal data in evaluating the likelihood that an exposure may cause a particular effect?
A. Show in me the animal data, and I'll tell you my opinion.
Q. All right. It may or may not be important, depending upon the circumstances?
A. Depending on the constructor of the study, depending on, you know, a variety of variables, it may or may not.
Q. Is it fair to say that animal models have been considered quite important in terms of evaluating the effect of many pharmacologic drugs?

A. Certainly, as a global statement.
Q. And if a person were to heavily weight the -- the results of animal experiments in their model of causation, would you have any quarrel with that?
A. It depends on the experiment, experimental design, what was proposed, the result of the study, the size of the study. I can't answer that as a general question. I mean, it depends on what the data is. I mean, garbage in equals garbage out. So, you know, I can do -- I can design a study that would purport to say something, but it may not say anything at all.
Q. How about -- how about a study -- after a well-constructed study that failed to demonstrate an animal model for a postulated human effect? Would that be --
A. Being as how the animal placenta, with the exception of primates, is considerably different than the human placenta, any placental work done on animals would be essentially worthless.
Q. So the comparability of the organ structure is incorrect?

A. For example, a piglet eye is very similar to the human eye, and so a lot of eye-type studies are done in piglets. However, the Beagle puppy brain appears to be similar to the premature human brain in some of its dynamics and bleeding tendencies, but we don't use a piglet brain because it doesn't look the same and it doesn't act the same. So you have to try to use comparable organ structures and comparable physiologic systems in order to make consequential conclusions.

Q. In terms of the relationship between maternal smoking and any of the adverse pregnancy outcomes that you have mentioned in Exhibit 6, would it be fair to say that you have not made any attempt to determine how likely smoking may have been in terms of producing any particular cause in the Texas Medicaid population?

A. Correct.

Q. Would it be fair to say that in the studies that you have reviewed which have associated maternal smoking and an adverse pregnancy outcome, that the -- the strength of the relationship between maternal smoking and

that outcome has varied from study to study?
A. Depending on the outcome you're looking at, depending on the size of the study, depending on the study design, one would anticipate there would be variability between studies.
Q. Does the term "relative risk" have meaning to you?
A. If you're talking about confidence intervals and that sort of thing, yes.
Q. All right. There -- generally, if a relative risk is a product of multiple regression analysis, you're going to expect to see that relative risk accompanied by a confidence interval --
A. Correct.
Q. -- correct? And would it be fair to say that what that means is that in the population that we have sampled, we can be 95 percent sure that that relative risk lies between "X" and "Y," "X" and "Y" being the upper and lower boundaries of the confidence internal?
A. If your "P" value is .05, that's what it says.
Q. "P" .05 or less if that's what you've said is your --

A. Well, no. If it's "P" value is .01, then you have a 99 percent chance of the fact being true within those confidence limits. And if you have a "P" value of .001, you have 999, I think, out of a thousand.
Q. You're exactly right.
A. So define your terms.
Q. Okay. If we have set "P" as .05, then what that confidence interval means is that, although we've given you a relative risk number which is at the mean or the median from within the data set that we've studied, what we're really telling you is that we can be 95 percent sure that the true relative risk lies between the lower boundary of the confidence interval and the upper boundary of the confidence interval, correct?
A. Usually one or two standard deviations, correct.
Q. And for any of the health effects that you're testifying about, do you know what the relative risk of maternal smoking is for that health effect as defined by the lowest low in a confidence interval that was found and the highest high?

A. No. Because, as I've stated earlier, I did not do a literature search in that regard.
Q. Did you do a search for the nonsmoking risk factor -- we talked about some nonsmoking risk factors for premature delivery or prematurity.
A. Correct.
Q. And we've mentioned a few with respect to low birth weight babies. Did you do any sort of search for the nonsmoking risk factors that apply to each of the health endpoints that you mentioned in Exhibit 6?
A. I did no literature searches, period.
Q. All right. Would you be prepared to tell us what the -- the nonsmoking risk factors were for each of the health endpoints that you've mentioned?
A. Today?
Q. Yes.
A. Given the limitations of my memory, I'll try my best.
Q. All right. What are the -- what are the nonsmoking risk factors for spontaneous abortion?
A. Chromosomal abnormalities, placental

abnormalities, infectious agents, smoking, drug abuse with cocaine. Depending on when the abortion occurs, potentially cervical incompetence.

Q. Are there any socioeconomic or sociodemographic risk factors for spontaneous abortions?

A. Youth.

Q. Low socioeconomic status?

A. I don't know.

Q. Are there differences by racial group?

A. There may be. But again, I'm not an obstetrician, and I am reporting to you what has been taught to me regarding abortion and the specific instance of tobacco use on the part of the mother. Investigation of various causes of abortion really are not germane to my practice.

Q. And I believe you pointed out to us earlier -- and if I got this wrong, say so -- it's typically not part of your practice to determine what of several risk factors may have contributed to or caused a particular effect; your job is to treat that condition as it's presented to you in that baby,

correct?
A. Simplistically, yes. Although, as I also mentioned earlier, I'm very interested in things that affect babies, because I may need to look at other problems if I know about other risk factors.
Q. Okay. So with respect to problems which may yet occur in babies or complicate conditions that they have, it may be an interest?
A. Well, for example, it's very valuable to me if I know a mother has diabetes. Okay? Because that is going to cause certain predictable problems in a baby. It is of interest to me that mothers are snorting cocaine at the time of delivery because that also can cause acute problems in the baby. It is of interest to me if she has infections that are ongoing because that is going to affect the welfare of the baby. So depending on the maternal cause and problem, it may have a great deal of effect on my treatment of the infant. So it's nice to have histories.
Q. All right. You gave us a list of risk factors or nonsmoking risk factors relating

to spontaneous abortions. Would you expect that the relative prevalence or mix of those risk factors could change from population to population, depending upon which population you looked at?

A. As a nice global statement, certainly.

Q. Okay. And if one were to investigate the potential role, for instance, of maternal smoking and spontaneous abortions in the Texas Medicaid population, wouldn't you expect that one would want to know what the prevalence or incidence of chromosomal abnormalities, placental abnormalities, infectious agents, smoking, drug abuse, cocaine, cervical incompetence, all the things that you mentioned, wouldn't you want to know that the relative prevalence of those risk factors were in that same population?

A. If the prevalence of each is similar on both sides of the equation, then they cancel each other out.

Q. Okay. I did, unfortunately, mention smoking on either side. But other --

A. You mentioned smoking on one side and other things in both. At least that was my

interpretation.
Q. Well, the -- if one were going to study the impact of a particular risk factor in a population, one would need to know the prevalence of other risk factors in that population, correct?
A. As a general statement.
Q. Because, otherwise, any potential association with the risk factor under study would be confounded by failure to control for the other risk factors, correct?
A. Upon occasion, as I pointed out earlier, if you have a large enough sample size, you tend to get away from such biases.
Q. All right. But different populations can have similar incidences of conditions for dramatically different reasons, correct?
A. Come again?
Q. Different populations can have similar incidences of a particular health endpoint for dramatically different reasons, can they not?
A. Getting back to the never nevers and never alwayses, certainly.
Q. All right. For instance, if one were to

investigate the incidence of low birth weight babies and one compared a population which was predominantly made up of white smoking women of high socioeconomic status and compared it to black nonsmoking women of low socioeconomic status, one might expect to find a fairly similar rate of low birth weight outcome between the two populations but for dramatically different potential reasons?

A. Potentially.

Q. And so would it not be correct to say that with respect to any of the health endpoints that you have in your disclosure statement, in order to attempt to ascertain the relative importance of maternal smoking in terms of producing or possibly producing that health endpoint in the Texas Medicaid population, one would want to know the relative prevalence of the other risk factors for those same disease endpoints in that population?

A. Again, if you have a large enough population and you're taking the entire Medicaid population of Texas and you're comparing it

with other folks, maybe or maybe not, because you may have washed out all the differences with the exception of smoking.

Q. Okay.

A. Small studies get you into trouble.

Q. All right.

A. Big studies help you out.

Q. All right. Do you have any idea of the variability, for instance, and the relative risk that has been determined in -- in large studies of maternal smoking and low birth weight outcome?

A. I think you asked that before.

Q. Well, do they vary by as much as a hundred percent?

A. I have not, as I keep telling you in response to your questions, done a literature search in these issues. So I'm not prepared to answer your question with any knowledge.

May I pick up my page?

Q. Absolutely. Do you want to take a break?

A. Please.

THE VIDEOGRAPHER: The time is 4:45 p.m. We're going off the Record.

(A recess was taken)

THE VIDEOGRAPHER: The time is 4:51 p.m. We're on the Record.

Q. (By Mr. Minton) Dr. Speer, just some general questions back about statistical associations in epidemiology.

Is it a statement of truth that because epidemiologic studies are based upon probability theory, that the statistical associations that they put forth apply to groups and not necessarily specific individuals among those groups?

A. That's true really of any finding, not just epidemiologic studies. Because in any study, you are measuring groups, and you are confirming or rejecting a null hypothesis. And it is unusual for any intervention to be a hundred percent negative or positive.

Q. Does it follow, then, that epidemiologic principles do not of themselves predict individual outcomes?

A. I think that's probably a true statement. An example, perhaps, might clarify it. We know that babies under the 1500-gram birth weight have an incidence of interventricular hemorrhage from 28 to 45 percent, depending

on the institution that reports. However, a given baby with an interventricular hemorrhage has a hundred percent chance of an interventricular hemorrhage, and obverse holds true also.

Q. And so the predictive capability in an individual case using epidemiologic data can be quite limited?

A. True. But for populations, it can be quite precise.

Q. The --

A. And if you want to flip coins and take chances, then that's what epidemiologic data allows you to do.

Q. All right. And using the coin flipping analogy, if we were to flip a fair coin with a fair person tossing it, we would expect to see an outcome of 50 percent heads and 50 percent tails, correct?

A. If you flip it long enough, yes.

Q. All right. But the epidemi -- or the statistics underlying that analysis won't tell us what the next toss is going to be, correct?

A. No. Each toss is a 50/50 chance.

Q. And -- and the same holds true in terms of the attempted application of epidemiologic data to an individual. If you attempt to apply epidemiologic data to an individual, you have an unknown or an unquantifiable rate of error?
A. However, if you have a 50 percent chance of getting something, then I would think that those risks would be far less attractive than a 1 percent chance of getting something.
Q. But -- but the original part of the question or the statement in that part of the question holds true, that if you attempted to apply epidemiologic data to an individual, you have an unknown or unquantifiable rate of error?
A. You -- depending upon the data that's available, the relative risk to that person would be enhanced, neutral or diminished, depending upon what the exposure and what you were measuring. So if you have a relative risk of twofold for a given condition, say, prematurity if you smoke, then you have twice the risk of having a premature baby as opposed to the population who doesn't smoke.
Q. Well, but -- but --

A. And if you want to take that risk on, then fine.
Q. But I thought the point you made earlier was, and relative risk is certainly illustrative of that point, that relative risk for a particular individual is not 2.0. It is either zero or 1, correct? It's a binary relationship when it comes down to a particular individual. It's either going to cause it, or it isn't.
A. Right. But if you have a risk of getting it that's twice what the general population is, then your risks are higher.
Q. All right. However, a particular individual's risk, because that person is an individual with an individual makeup, is not necessarily and probably almost certainly not the same as the median risk that is derived from a population measure, correct?
A. The risk to a given individual of an event occurring, assuming that the data that you have is complete and well-done, ranges between your confidence limits around a mean.
Q. The risk for a particular individual is either zero or 1, isn't it?

A. No, no, no. You're saying if -- you're taking an individual, and you're saying, "Okay, does he have it, or he doesn't have it?" But the risks of getting it are what I'm talking about. You're talking about the risks of having it are zero to 1. I'm talking about the risks of getting it, and they're two different species.
Q. All right. But he's either going to get it or he's not going to get it, right?
A. But the risk of getting it may be twofold or threefold or fivefold or a hundredfold worse than the other individual who has not got the risk factor.
Q. Right. But a population risk factor becomes irrelevant in the individual circumstance, does it not?
A. No. As I've just stated, it can't, not if the relative risk is higher than the population at risk.
Q. All right. Do you know how one calculates the predictive capacity to an individual from a relative risk?
A. Not sitting here today, no. But I can probably find out.

Q. Would it be fair to say that in order to make a judgment about causation in an individual case, you have to have knowledge about that specific individual, the risk factors that that specific individual encountered, at what time they encountered them, how much of that risk factor they encountered, how that disease presented itself, all that data being unique to that particular individual?
A. It's not necessarily unique to that particular individual. As I've already just stated, if the risk associated with a given exposure is greater than that risk that the general population enjoys not being exposed to that agent, the risk to the individual at risk is higher.
Q. All right. But if we made a judgment of causation simply on the basis of that perceived increased risk from epidemiologic studies, we would have an unknown or unquantifiable rate of error?
A. A risk is a cause, and it is a risk. I mean, it's pretty simple. I mean, either you have a risk, or you don't have a risk. If you have a risk, then you're at risk. Right?

I'll tell you what, I'll let you cogitate on that answer. And why don't we resume tomorrow?

Q. Okay.

(Speer Exhibit No. 12 marked for identification)

MR. BLEVINS: Briefly on the Record, if we could go ahead and identify that Exhibit 12 is the news release and press statements by the American Academy of Pediatrics previously utilized in the doctor's testimony.

MR. MINTON: Thank you.

THE VIDEOGRAPHER: It's 5:01 p.m. We're going off the Record.

DEPOSITION OF MICHAEL SPEER, M.D.

CHANGE/CORRECTION PAGE

Please indicate changes on this sheet of paper, giving the page and line number, the change and the reason for the changes. Reason for changes are: (1) To clarify the record; (2) To conform to the facts; (3) To correct transcription errors.

PAGE/LINE	CORRECTION	REASON

SIGNATURE OF WITNESS

I have read the foregoing transcript of my deposition taken on the 3rd day of September, 1997, and it is a true and accurate record of my testimony given at that time and place, except as to any corrections I have listed on Page 240 herein.

_____
MICHAEL SPEER, M.D.

THE STATE OF TEXAS)
COUNTY OF HARRIS)

SUBSCRIBED AND SWORN TO BEFORE ME, the undersigned authority, on this the _____ day of _____, 19____.

_____
NOTARY PUBLIC IN AND FOR
THE STATE OF T E X A S

My Commission Expires:
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STATE OF TEXAS)
COUNTY OF HARRIS)

REPORTER'S CERTIFICATION

DEPOSITION OF MICHAEL SPEER, M.D.

TAKEN SEPTEMBER 3, 1997

I, LETTIE WITTER, Certified Shorthand Reporter for the State of Texas, hereby certify that this deposition transcript is a true record of the testimony given by the witness named herein, after said witness was duly sworn by me.

I further certify that I am neither attorney nor counsel for, related to, nor employed by any of the parties to the action in which this testimony is taken. Further, I am not a relative nor employee of any attorney of record in this cause, nor do I have a financial interest in this action. Further certification requirements pursuant to the Rules will be certified to after they have occurred.

Subscribed and sworn to on this the ______ day of __________________, 1997.

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IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TEXAS
TEXARKANA DIVISION

THE STATE OF TEXAS, Plaintiff VS. THE AMERICAN TOBACCO COMPANY; R.J. REYNOLDS TOBACCO COMPANY; BROWN & WILLIAMSON TOBACCO CORPORATION; B.A.T. INDUSTRIES, P.L.C.; PHILIP MORRIS, INC.; LIGGETT GROUP, INC.; LORILLARD TOBACCO COMPANY, INC.; UNITED STATES TOBACCO COMPANY; HILL & KNOWLTON, INC.; THE COUNCIL FOR TOBACCO RESEARCH-USA, INC. (Successor to Tobacco Institute Research Committee); and THE TOBACCO INSTITUTE, INC., Defendants	CIVIL ACTION NO. 5-96CV91 UNITED STATES JUDGE: DAVID FOLSOM UNITED STATES MAGISTRATE: WENDELL C. RADFORD

CERTIFICATE TO THE DEPOSITION OF
MICHAEL SPEER, M.D.
TAKEN ON SEPTEMBER 3, 1997

I, LETTIE WITTER, a Certified Shorthand Reporter in and for the State of Texas, hereby certify pursuant to the Rules and/or agreement of the parties present to the following:
That this deposition transcript is a true record of the testimony given by the witness named herein, after said witness was duly sworn by me;
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MR. MICHAEL MINTON, Attorney for Defendants, Lorillard Tobacco Company:
MR. BRYAN BLEVINS, Attorney for Plaintiff;

That a copy of this certificate was served on all parties shown herein.

Given under my hand and seal of office on this the ____ day of _____, 1997.

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